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AN INTERESTING EXPERIMENT

It has been announced that Livingston County, Michigan, is to be the locale for an interesting experiment to be conducted under the guidance of the Coöperative and Community Service Section of the Resettlement Administration, and the Extension Department of Michigan State College. Stated very briefly, the object of the experiment is to try out a plan whereby low-income farmers may obtain competent veterinary service at a minimum cost. The proposed plan is modeled after the Missco Community Medical Association and calls for the formation of an organization which will contract with one or more veterinarians located in Livingston County to render certain services for a fixed fee.

Although the proposed plan has many of the earmarks of group medicine, and many will be inclined to oppose it on that score, we have tried to pick out what would appear to be desirable features of the experiment. The plan does not differ materially from so-called contract practice, which was very much in vogue in every one of our large cities a generation ago, when horses were used almost exclusively to supply vehicular power. In those days, a veterinarian did not hesitate to take a large barn on an

annual contract basis and render all necessary veterinary services for so much per horse per year.

The Livingston County plan provides that the veterinarian will make one visit per month to the farm of each member of the association and render all of the veterinary services, including dehorning, castrating, docking, vaccinating, and giving advice on such matters as feeds and feeding. In addition to this, the veterinarian will make two emergency calls per year without extra charge. Additional emergency calls will be rendered at a stipulated rate. It is proposed that the veterinarian furnish all necessary medicine except biological products and expensive pharmaceutical preparations, these to be furnished at cost.

On the basis of 70 R. A. clients who are prospective members of the association, it is planned to render this veterinary service at the following cost to each client: a $5.00 membership fee and, in addition, a fee of 80 cents per year per productive animal unit. The productive animal unit used by the Michigan State College Farm Management Division consists of the following: 1 horse, 1 cow, 2 young cattle, 5 hogs, 7 sheep, or 100 poultry. For example, a client who has 2 horses, 4 cows, 2 heifers, 2 brood sows and their litters, and 100 poultry could be a member of an association and receive the veterinary service stipulated at an annual cost of $13.00. On this basis, 70 clients would pay $910 per year. The plan provides that the veterinarian receive his remuneration in 12 monthly installments.

It is reported that all veterinarians who have been approached with the proposition are in favor of the plan, as it will yield a sure, steady income for approximately six work days each month, with 275 miles of travel on regular visits, plus whatever emergency calls may be necessary. It is believed that the regular monthly visits will keep down the number of emergency calls, which is one of the many objects of the scheme.

One decided advantage of the plan is to be found in the fact that the veterinarian will be called upon to render services which ordinarily are not performed by the veterinarian. We have in mind such operations as castrating and docking and, to a certain extent, dehorning and vaccinating. Many of these operations are performed right along by farmers themselves.

A tremendous advantage is to be found in that part of the plan which provides for the veterinarian acting as an advisor in animal husbandry problems, including feeds and feeding, breeds and breeding, and all of those activities which come under the general head of animal management. We know of nothing that will serve to bring the veterinarian and the stockman closer

together than this advisory relationship. The farmer has been in the habit of going elsewhere for information, mainly for the reason that there have been places where he could get veterinary advice without having to pay a direct fee for it.

The salary of the veterinarian paid by the association is practically guaranteed. There will be no problem of unpaid veterinary bills, and there will be practically no chance for an argument over charges made by the veterinarian. These will be fixed in advance.

The periodic visits of the veterinarian to each farm should prove valuable in preventing the development of disease. At the present time, we have practically nothing in the form of regular farm inspection by veterinarians for the purpose of detecting diseases in their incipiency. The Resettlement Administration recognizes the benefits to be derived from this form of veterinary protection to the live stock which is a part of the security for the loans made to clients.

At the recent meeting of the Michigan State Veterinary Medical Association, the plan was discussed very thoroughly and was referred to a committee for study. It is realized that there may be some wrinkles in the proposed setup which will have to be ironed out, but there appear to be no very serious obstacles in the way. The experiment will be watched with considerable interest, not only by Michigan veterinarians, but by those in other states.

ADD NEBRASKA

On June 1, Nebraska was designated as a modified accredited area by the U. S. Department of Agriculture, bringing the number of states practically free from bovine tuberculosis up to 40. Although testing has been in progress in Nebraska for a number of years, most of the progress toward the coveted goal has been made within the past two years.

The accrediting of Nebraska means much to the live stock industry of that state on account of the large number of feeder cattle that are shipped out of the state, and which may now enter interstate commerce without the tuberculin test.

Things have been going the wrong way in the adjoining state of South Dakota, if we are to judge from press dispatches of June 24, stating that the South Dakota Supreme Court had ruled that the compulsory testing of cattle for tuberculosis could not be enforced under the existing laws of the state. Numerous injunctions have been issued to restrain the state and federal live stock sanitary officials from proceeding with the work of testing,

and now, as a result of the decision of the Supreme Court, the work will be held up in about a dozen counties.

Without in any way questioning the decision of the Supreme Court, we do feel sorry for the misguided cattle-owners in South Dakota who have seen fit to invoke legal proceedings to hinder the work of eradicating tuberculosis from their herds, something which has been initiated largely in their own interests. To use a well-known expression, these South Dakota cattle-owners "have only been biting off their own noses to spite their faces." Their action will merely delay the accomplishment of the end sought and, in the meantime, these misguided farmers will be the losers through having the markets for their cattle and dairy products very much restricted.

Besides South Dakota, there remain seven other states—California, New York, New Jersey, Pennsylvania, Maryland, Vermont and Rhode Island—that are not yet accredited.

EXECUTIVE BOARD ELECTIONS

As a result of the regular quintennial Executive Board elections which came to a close on June 13, Dr. I. E. Newsom, of Fort Collins, Colorado, was elected in District 6 (California, Nevada, Utah, Colorado, New Mexico, Arizona, Mexico, Central America and Canal Zone) and Dr. L. J. Allen, of Oklahoma City, Oklahoma, was elected in District 8 (Kansas, Missouri, Oklahoma, Arkansas, Texas and Louisiana). The terms of Drs. Newsom and Allen will start at the close of the meeting in Columbus.

DESHLER-WALLICK HOTEL

Columbus Hotels

Hotel	Address	Distance from Headquarters (blocks)	Rooms	Rates			
				Without Bath		With Bath	
				Single	Double	Single	Double
Deshler-Wallick* (Official Headquarters)	Broad and High Sts.	Headquarters	1000	.....	.....	$2.50-$8.00	$4.50-$9.00 Twin beds, $5.00-$10.00 Suites, $8.00-$18.00
Neil House	Opposite State Capitol Bldg., High St.	½ South	655	.....	.....	$2.50-$4.50 Suites, $7.00 up	$3.50-$5.50 Twin beds, $5.00-$7.00 Suites, $10.00 up
Fort Hayes†	West Spring St.	4½ North	300	.....	.....	$2.50-$3.50 Suites, $6.00-$8.00	$4.00-$6.00 Suites, $8.00-$10.00
Chittenden	Spring and High Sts.	4½ North	300	$1.50-$2.00	$2.50-$3.00 Twin beds, $3.50	$2.50-$3.00	$3.00-$4.00 Twin beds, $4.50-$6.00 Additional person, $1.50
Southern‡	Main and High Sts.	5 South	230	$1.50-$1.75	$2.50-$2.75	$2.00-$2.50	$3.00-$3.50 Twin beds, $4.00-$4.50
Virginia	Third and Gay Sts.	1 North and 1 East	150	$1.50	$2.00-$2.50	$1.75-$2.50	$3.00-$4.00
Jefferson	East Spring St.	4½ North	75	$1.25	$2.00-$2.50	$2.00	$3.00-$3.50

*Large capacity rooms accommodating 4 to 10, $1.50-$2.50 per person.
†Large capacity rooms accommodating 4 to 15, $1.00-$1.50 per person.
‡Large capacity rooms accommodating 4 or more, $1.50 per person.

ON TO COLUMBUS

Columbus, the A. V. M. A. convention city of 1936, is not only a modern metropolis of great activity, but is a state capital of scenic beauty with wide streets, beautiful drives and well-kept parks, situated along the Scioto and Olentangy rivers. The city administrators have not only provided for permanent public welfare, but have actively promoted a comprehensive recreational program. There are golf courses, bridle paths, and a river boulevard which extends twenty-one miles up the picturesque Scioto River, and the eleven miles of delightful picnic grounds along the river are among the finest contributions any city has made to its people. In addition, boating and swimming are offered on two large lakes within easy motoring distance.

Visiting members of the A. V. M. A. will find in Columbus an uncrowded and uncongested urban center with an excellent transportation system which greatly facilitates traveling to any desired point. Hotels found here are unsurpassed by those of any other city of its size. The Deshler-Wallick will be official headquarters and the management is prepared to extend every hospitality and as efficient, courteous service as anyone may desire. As the opening date of the convention, August 11, is only a little over a month away, it would be advisable to make your reservations now. A table showing the names and rates of the principal Columbus hotels appears on the opposite page.

On to Columbus!

THE CONVENTION PROGRAM

As a result of the splendid coöperation rendered by the officers of the five sections of the A. V. M. A., we are able to present, more than a month in advance of the meeting, a practically complete list of the papers that will be presented at the Columbus convention. It would hardly be fair to single out the program of any one section and say anything about the importance of the subjects to be presented before that section. There is not a subject on the list of papers that is not important to somebody or some group. However, we would like to direct the attention of the practitioners particularly to the papers to be presented before the two practice sections. The Section on General Practice will take care of horses, cattle, sheep and swine, and the Section on Small Animals will look after the interests of the dog in particular. An effort is being made to schedule the two meetings of both sections at times when they will not conflict. In this way

practitioners will have an opportunity to hear all papers read before both sections.

Another feature of the program, one that is just a bit novel, to say the least, is the session of the Section on Sanitary Science and Food Hygiene that is to be devoted entirely to meat and milk inspection, a field that is becoming increasingly important to the veterinarian every day. Poultry diseases and problems will receive their share of attention in the Section on Poultry and the laboratory men will find a literary feast in the program of the Section on Research.

The final day of the meeting, Friday, August 14, will be given over entirely to a clinic, which will be held in one of the buildings on the Ohio State Fair grounds, a ten-minute drive from the convention headquarters. The clinic will be directed by Dr. W. F. Guard, of Ohio State University, and no detail will be overlooked by Dr. Guard and his capable committee to make the clinic one of the most complete ever held.

The list of papers (tentative) follows:

General Session

The American Stockman and the Veterinary Profession—Mr. Samuel R. Guard, Editor, Breeder's Gazette, Spencer, Ind.

Tularemia (Illustrated)—Dr. Walter Simpson, Miami Valley Hospital, Dayton, Ohio.

Progress Report on Crystal-Violet Vaccine Against Hog Cholera—Dr. C. N. McBryde, U. S. Bureau of Animal Industry, Ames, Iowa.

Studies on Gastric Digestion in Ruminants (Illustrated)—Dr. A. F. Schalk, Ohio State University, Columbus, Ohio.

Section on General Practice

Control of Parasites in Sheep—Dr. J. H. Rietz, West Virginia University, Morgantown, W. Va.

The General Practitioner—Dr. T. A. Sigler, Greencastle, Ind.

Diseases of Sheep—Dr. F. E. Hull, University of Kentucky, Lexington, Ky.

Swine Diseases—Dr. John B. Bryant, Mount Vernon, Iowa.

Arthritis of the Horse—Dr. Cassius Way, New York, N. Y.

Mastitis in Cattle—Dr. C. H. Case, Akron, Ohio.

Anaplasmosis of Cattle—Dr. Hubert Schmidt, Texas Agricultural Experiment Station, College Station, Texas.

Pyelonephritis in Cattle and Horses—Dr. W. L. Boyd, University of Minnesota, Saint Paul, Minn.

Section on Sanitary Science and Food Hygiene

Live Stock Loss Prevention—Mr. L. L. Rummel, Secretary, Ohio Live Stock Loss Prevention Association, Columbus, Ohio. Discussion by Dr. W. J. Embree, New York Central Lines, Columbus, Ohio, and Dr. W. T. Spencer, Live Stock Commissioner, Omaha, Neb.

Regulating the Sale and Use of Biological Products Containing Pathogenic Organisms—Dr. H. M. Kalodner, Director, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.

Paper—Dr. H. E. Moskey or Mr. S. A. Postle, Food and Drug Administration, U. S. Department of Agriculture, Washington, D. C.

Auction and Community Sales—Dr. F. A. Zimmer, State Veterinarian, Ohio Department of Agriculture, Columbus, Ohio.

Infections Transmitted in Milk—Dr. J. C. Wickham, Cleveland, Ohio.

Meat Inspection—Dr. Warren P. S. Hall, Division of Health, Toledo, Ohio.

Report of Special Committee on Meat Hygiene:

(a) Municipal Meat Inspection—Dr. E. E. Chase, Chief Meat Inspector, Bureau of Health, Portland, Ore.

(b) State Meat Inspection—Dr. E. T. Faulder, Director, Animal Industry Bureau, Department of Agriculture and Markets, Albany, N. Y.

(c) Poultry Inspection—Dr. Colonel E. Edmunds, Supervising Inspector, Bureau of Agricultural Economics, Chicago, Ill.

(d) What Our Veterinary Colleges Are Doing to Prepare Veterinarians for Public Health Work—Dr. Ward Giltner, Dean, Division of Veterinary Medicine, Michigan State College, East Lansing, Mich.

(e) Recommendations of the Special Committee on Meat Hygiene—Dr. J. S. Koen, Chairman.

(f) What Canada Is Doing Along Meat Inspection Lines—

Section on Research

Studies on Bovine Blood. The Sedimentation Rate and Percentage Volume of Erythrocytes in Normal Blood—Lloyd C. Ferguson, University of Wisconsin, Madison, Wis.

The Pathogenesis of Ketosis. Pregnancy Disease of Sheep—Drs. Lee M. Roderick, G. S. Harshfield and M. C. Hawn, North Dakota Agricultural Experiment Station, Fargo, N. Dak.

The Significance of Suspicious Agglutination Reactions for Bang's Disease—Dr. C. P. Fitch, Lucille M. Bishop and Dr. W. L. Boyd, University of Minnesota, University Farm, Saint Paul, Minn.

The Significance of Pasteurella Boviseptica Encountered in the Blood of Some Florida Cattle—Dr. D. A. Sanders, Florida Agricultural Experiment Station, Gainesville, Fla.

The Problem of the Vectors of Anaplasmosis in the United States —G. Dikmans, United States Bureau of Animal Industry, Washington, D. C.

Infectious Diseases of Domestic Rabbits—Drs. J. E. Shillinger and F. D. McKenney, Bureau of Biological Survey, U. S. Department of Agriculture, Washington, D. C.

Section on Small Animals

A Consideration of Heartworm and Tapeworm Infestations in the Dog—Dr. R. E. Rebrassier, Ohio State University, Columbus, Ohio.

The Prostate Gland of the Dog—Dr. C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.

The Diagnosis, Prognosis and Treatment for Hip Injury—Dr. E. F. Schroeder, Boston, Mass.

A Study of Streptococcic Infections in Dogs—Dr. H. J. Stafseth, Michigan State College, East Lansing, Mich.

Some of the Pathological Lesions Associated with Indigestion in Dogs—Dr. Leonard W. Goss, Ohio State University, Columbus, Ohio.

Observations on Canine Piroplasmosis—Dr. D. A. Sanders, Florida Agricultural Experiment Station, Gainesville, Fla., and Dr. J. H. Yarborough, Miami, Fla.

The Utilization of Sheep in the Rabies Vaccine Protection Tests —Dr. J. E. Schneider, Mulford Biological Laboratories, Glenolden, Pa.

Section on Poultry

Infectious Enterohepatitis—Dr. A. J. Durant, University of Missouri, Columbia, Mo.

Salmonella Infections in Poultry—Dr. Henry Van Roekel, Massachusetts State College, Amherst, Mass.

Studies on the Egg Propagation of Laryngotracheitis and Fowl-Pox—Dr. C. A. Brandly, Kansas State College, Manhattan, Kan.

Relative Values of Farm Grains in Poultry Nutrition—Dr. H. L. Wilcke, Iowa State College, Ames, Iowa.

The Results of the Practical Application of Pullorum Disease Control Methods—Dr. J. T. Burriss, Ohio Department of Agriculture, Reynoldsburg, Ohio.

Fowl Leukosis—Dr. C. D. Lee, Iowa State College, Ames, Iowa.

Field Observations on the Incidence of Fowl Leukosis in Hatching Eggs and Chicks—Dr. B. A. Beach, University of Wisconsin, Madison, Wis.

The Cultivation of the Virus of Chick Bronchitis—Dr. F. R. Beaudette, New Jersey Agricultural Experiment Station, New Brunswick, N. J.

The Importance of Endotoxin in the Moderation of Fowl Paralysis by Bacteria of the Paratyphoid Group—Dr. M. W. Emmel, Florida Agricultural Experiment Station, Gainesville, Fla.

No Railroad Rates to Columbus

As a result of the reductions in railroad fares which recently went into effect, there will be no special convention rates to Columbus. We are informed that the certificate plan, which has been in use for a number of years, has been discontinued entirely. This notice is being published at this time to forestall the inquiries which are received every year relative to special railroad rates to the convention city.

New State Office Building, Columbus

ENTERTAINMENT FEATURES

The ladies and children attending the Columbus meeting are going to be taken care of in grand style, if we may judge from the preparations being made for their entertainment by Dr. and Mrs. O. V. Brumley, who head the committee in charge of the various entertainment and social features of the week. Assisting Dr. and Mrs. Brumley will be the following Columbus veterinarians and their wives:

Dr. and Mrs. D. W. Ashcraft, Dr. and Mrs. H. G. Bond, Dr. and Mrs. W. A. Brown, Dr. and Mrs. L. W. Goss, Dr. and Mrs. Jas. D. Grossman, Dr. and Mrs. W. F. Guard, Dr. and Mrs. D. C. Hyde, Dr. and Mrs. J. W. Jackman, Dr. and Mrs. W. R. Krill, Dr. and Mrs. George Mechling, Dr. and Mrs. E. P. Maxwell, Dr. and Mrs. R. E. Rebrassier, Dr. and Mrs. A. F. Schalk and Dr. and Mrs. F. A. Zimmer.

In another section of the preview of the program will be found an outline of the entertainment planned for the children. The following program has been tentatively arranged for the ladies. They will attend the opening session on Tuesday morning and a theater party in the afternoon. In the evening they will attend the alumni banquets and the President's reception and dance.

On Wednesday there will be a trip to the Columbus Country Club, where luncheon will be served. The convention banquet is scheduled for the evening. On Thursday the annual meeting of the Ladies' Auxiliary to the A. V. M. A. will be held. A party and luncheon will occupy Friday.

BRING THE CHILDREN

Extraordinary plans are being made by the Committee on Local Arrangements, at Columbus, to provide for the care and entertainment of the children whose parents will attend the convention. The special committee, headed by Mrs. James D. Grossman, has planned a variety of entertainment for the children during the week.

On Tuesday afternoon there will be a theater party and another one that evening. This will make its possible for the ladies to attend the alumni banquet Tuesday evening if they so desire. On Wednesday morning, the children will be transported to the municipally owned Shutter House on the beautiful Scioto River where they will enjoy an all-day picnic and outing until 4:00 p. m. Wednesday evening while their parents are attend-

ing the convention banquet, the children will be given another theater party.

On Thursday, the children will be entertained at Olentangy Park from 10 a. m. until 4:00 p. m. A free luncheon will be provided for the children, as well as free tickets that will be good for anything in the park except food. On Friday, there will be a trip to the penitentiary, the campus of Ohio State University and the museum. Before leaving the campus, luncheon will be served at Poamerine Hall, following which the children will be returned to Goodale Park where they will remain until 2:30 p. m.

In following the above schedule, the children will be under the supervision of Miss Maida Shore and her eight assistants. Miss Shore is recreational director of the First Community Church at Greenview, Ohio, and is well trained and adapted for the responsibility which will be placed upon her.

Bring the children!

EXHIBITS

The entire third floor of the Deshler-Wallick Hotel will be given over to the exhibits. According to the latest information received from Dr. E. P. Maxwell, chairman of the Committee on Exhibits, the sale of space has been very encouraging and practically every veterinary supply house in the United States is expected to answer the roll-call of exhibitors at Columbus. At no other time during the year and at no other place than an A. V. M. A. convention will be found such an array of new instruments and equipment, improved pharmaceutical and biological products, in fact everything the veterinarian uses in going about his work each day. The complete list of exhibitors will be published in the August issue of the JOURNAL.

PARKING FACILITIES IN COLUMBUS

Dr. A. J. DeFosset, chairman of the Committee on Transportation and Parking, supplies the following information relative to parking facilities in Columbus:

Merchants' Garage, 100 N. Front St., located about 1½ blocks west of the Deshler-Wallick Hotel, has space available for inside parking of 400 cars. Rate: "In and out service" for 24 hours, 75 cents per day.

Out-door parking lots within a range of three blocks of the hotel can accommodate 650 autos. The parking rates in these

out-door lots range from 5 to 50 cents for 24-hour service. Attendants or watchmen are not on duty at night in the cheaper parking lots.

On the State Fair Grounds, free space is available for 4,000 cars for the accommodation of those attending the clinic.

Dr. DeFosset has very thoroughly canvassed the touring camps in the vicinity of Columbus, particularly along Route 40, and the results of his investigations are being placed at the disposal of A. V. M. A. members in the accompanying table. Practically every camp listed is well located and has good facilities for accommodating guests. The table shows the location, size, rates and facilities of each camp.

TOURIST CAMPS NEAR COLUMBUS

NAME	LOCATION	CABINS	RATES	FACILITIES*
Majestic Camp (Ned Frank)	20 mi. east Route 40	7	Single $1.00 Double $1.50	T E C 35c
Willow Dale Camp (W. E. Morgan)	19 mi. east Route 40	6	Single $1.25 Double $2.00	T E C 35c
Shamrock Touring Camp† (E. Williams)	18 mi. east Route 40	16	Single $1.50 up Double $2.50	T E C 50c
Jimmie's Barbecue (Jas. Mrazek)	12 mi. east Route 40	2	Single $1.25 Double $2.50	T E 50c
Indian Camp (W. E. Davis)	5 mi. east Route 40	13	Single $1.25 up Double $2.00	T E C 50c
Big Darby Inn (J. L. Houk)	12 mi. west Route 40	2	Single $1.00 Double $2.00	
Ten Mile Inn (Bagley McGee)	10 mi. west Route 40	7	Single $1.25 up Double $2.50 up	T E C
Seven Mile Inn (J. Aleshenick)	7 mi. west Route 40	6	Single (3) $1.75 Double $2.00 up	T E 50c
Seven Acre Camp‡ (B. M. Alcorn)	6 mi. west Route 40	18	Single $1.00 up Double $2.00 up	T E C 50c
Thompson Service Station	8 mi. south Route 3	7	Single $1.25 Double $2.50	T E 50c
Eitel's Service Station	11 mi. south Route 3	3	Single $1.25	T E 50c
Pure Oil Station (C. B. Alkire)	1½ mi. south Parsons Ave.	1	Single $2.00	

*T, space available for trailers; E, electricity; C, cooking. Rate per night for trailer and electricity where available.
†Very nice.
‡Exceptionally good.

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

APPLICATIONS FOR MEMBERSHIP

This month we are giving first listing to 48 applications for membership, the largest number for any one month since September, 1931. Note that 34 of the applicants are 1936 graduates. For this we have two members to thank, Dr. E. E. Wegner, of the State College of Washington, and Dr. W. F. Guard, of Ohio State University, who assisted the members of the graduating classes in their respective institutions in preparing their applications for membership in the A. V. M. A.

The applications listed this month bring the total for the year (ten months, to be exact) up to 231. This figure exceeds the total of 220 for the full year 1934-35, and puts us in a position where we may be able to better the figure of 302 for the year 1929-30. One thing is certain and that is the fact that the annual report of the Secretary, to be presented at Columbus, next month, will show a nice increase in total membership as compared with one year ago.

As is our semi-annual custom, we are quoting Section 1 of Article 2 of the By-laws, covering the manner of filing an application for membership.

> Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

ARRON, DANIEL P. 312 32nd Ave., Seattle, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: Hilton A. Smith and E. E. Wegner.

BAUM, HARVEY 24 Maple St. S. E., Massillon, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

BERGER, GEORGE 18 Edmondson Ave., Lexington, Va.
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Leonard W. Goss.

BERLINER, MEYER 6 Margot Pl., Great Neck, L. I., N. Y.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

BORDNER, LAWSON A. 903 13th Ave. S., Nampa, Idaho
D. V. S., Grand Rapids Veterinary College, 1906
Vouchers: J. E. Ellis and T. W. White.

BRADLEY, DONALD H. 1569 N. High St., Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

BRAND, RUDOLPH W. 613 Lincoln St., Longmont, Colo.
D. V. M., Colorado State College, 1928
Vouchers: I. E. Newsom and A. A. Hermann.

CANTRALL, EMMETT W. Likely, Calif.
B. S., D. V. M., State College of Washington, 1936
Vouchers: Leonard L. Newman and Wm. C. Bellis.

CHRISTOPHER, BURTON C. 303 E. Union Ave., Olympia, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and N. G. Covington.

COLLINS, FLOYD M. Lyons, Neb.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

COOK, ROGER A. 205 N. Sprague Ave., Bellevue, Pittsburgh, Pa.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and J. H. Knapp.

COX, DANFORD L. Box 313, Wausau, Wis.
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Leonard W. Goss.

CROMLEY, CURTIS W. Ashville, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

DODGE, IDA MAE Harrison Ave. at Three-Mile, Butte, Mont.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

DODGE, ROGER E. Harrison Ave. at Three-Mile, Butte, Mont.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

DOUGHERTY, ROBERT W. Ohio State University, Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and J. H. Knapp.

FLEMING, JOHN Box 313, Wausau, Wis.
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Leonard W. Goss.

GEYER, HARRY G. Grove City, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

GREEN, KENNETH L. R. 4, Golden, Colo.
D. V. M., Colorado State College, 1931
Vouchers: James Farquharson and I. E. Newsom.

GUARD, WILLIAM P. Friday Harbor, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and Leonard L. Newman.

HACKETT, CLARENCE P. Kinsman, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and J. H. Knapp.

HAMMOND, WILLIAM H. R. 1, Monterey, Calif.
D. V. M., Ohio State University, 1926
Vouchers: W. L. Curtis and C. B. Outhier.

HENKEL, ERNEST L. 1606 Ruby St., Pullman, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and Hilton A. Smith.

HERMAN, LESLIE F. 8016 Whitethorne Ave., Cleveland, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

HOLMBERG, GERALD W. 45 W. 11th Ave., Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

HOUK, WILLIAM S. 402 Park Dr., Muscatine, Iowa
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

JONES, KENNETH S. Rushsylvania, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and J. H. Knapp.

JONESCHILD, WILLIAM R. 616 S. Adams, Tacoma, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and Leonard L. Newman.

JURDEN, RICHARD H. Box 3002, San Carlos, Calif.
D. V. M., Kansas State College, 1933
Vouchers: W. L. Curtis and Col. J. R. Shand.

KOUTZ, FLEETWOOD R. 204 Spaulding St., Pullman, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and Leonard L. Newman.

LEGGETT, WILLIAM R. 99 Windsor St., Waterbury, Conn.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

LONG, GERALD H. Mount Sterling, Ill.
D. V. M., Iowa State College, 1934
Vouchers: C. C. Hastings and W. B. Holmes.

MILLER, JAMES J. 1371 Fulton St., San Francisco, Calif.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and Leonard L. Newman.

MILLS, JOHN W. Ohio State University, Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

MOORE, MORRIS E. Flora, Ind.
D. V. M., Ohio State University, 1936
Vouchers: J. H. Knapp and W. F. Guard.

MOTTELER, GEORGE F. Colville, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: E. E. Wegner and Leonard L. Newman.

NIXON, GAYLORD J. R. 5, Mansfield, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

PALMER, CHARLES A. 610 W. Masonic St., Gainesville, Fla.
D. V. M., Iowa State College, 1920
Vouchers: D. A. Sanders and A. L. Shealy.

RICE, ALLEN W. 507 Federal Bldg., Little Rock, Ark.
D. V. M., Cincinnati Veterinary College, 1912
Vouchers: C. D. Stubbs and P. A. Johnson.

SPANGLER, HAROLD M. 1116 Bluemont Ave., Manhattan, Kan.
D. V. M., Ohio State University, 1935
Vouchers: E. E. Leasure and J. H. Burt.

STEPHAN, SOL G. 30 Erkenbrecher Ave., Cincinnati, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Leonard W. Goss.

TALCOTT, ROBERT V. P. 925 California Dr., Burlingame, Calif.
B. S., D. V. M., State College of Washington, 1936
Vouchers: J. K. Perry and E. E. Wegner.

TWISSELMANN, NORMAN M. Maricopa, Calif.
D. V. M., Iowa State College, 1936
Vouchers: Chas. Murray and C. H. Covault.

UTTERBACK, JOHN A., JR. Milner, Colo.
D. V. M., Colorado State College, 1934
Vouchers: James Farquharson and I. E. Newsom.

WAGERS, ROBERT P. Ohio State University, Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: A. F. Schalk and Leonard W. Goss.

WEIGAND, HARRY G. 2750 S. Broadway, Englewood, Colo.
D. V. M., Colorado State College, 1917
Vouchers: I. E. Newsom and A. A. Hermann.

WILLIAMS, GEORGE A. 831 W. Milford St., Glendale, Calif.
B. S., D. V. M., State College of Washington, 1936
Vouchers: P. H. Blickenstaff and G. W. Blanche.

WINIECKI, HENRY F. 702 Cherry St., Raymond, Wash.
B. S., D. V. M., State College of Washington, 1936
Vouchers: C. A. Johnston and N. G. Covington.

Applications Pending

SECOND LISTING

(See June, 1936, JOURNAL)

Carlin, Lt. Max H., 569 E. 106th St., Cleveland, Ohio.
Cottingham, Jay, 321 Federal Bldg., Salt Lake City, Utah.
Ferguson, Lloyd C., 607 W. Johnson St., Madison, Wis.
Koudelka, Thomas P., 1102 State Office Bldg., Richmond, Va.
Millenbruck, Lt. Edwin L., Fort Bragg, N. C.
Nowlin, Hubert A., University Station, Gainesville, Fla.
Orr, Norton A., Box 171, Fillmore, Utah.
Prentice, Carl M., 115 Duane St., Clyde, Ohio.
Pritchard, Clare W., 3315 N. 24th St., Tacoma, Wash.
Simonson, Earl M., Hooper, Neb.
Straker, Joe H., 201 E. Third St., Greenville, Ohio.
Tucker, Glenn A., Vista, Calif.

The amount which should accompany an application filed this month is $7.50, which covers membership fee and dues to January 1, 1937, including subscription to the JOURNAL.

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

COMING VETERINARY MEETINGS

Houston Veterinary Association. Houston, Texas. July 2, 1936. Dr. D. B. Strickler, Secretary, 317 Federal Bldg., Houston, Texas.

South Carolina Association of Veterinarians. Claremont Hotel, Sumter, S. C. July 6-7, 1936. Dr. R. A. Mays, Secretary, 408-410 State Office Bldg., Columbia, S. C.

Northwest Veterinary Medical Association. (Joint meeting of the Oregon, Washington and British Columbia Veterinary Medical associations.) Winthrop Hotel, Tacoma, Wash. July 6-8, 1936. Dr. Cliff. Ackley, Secretary, 1610 Market St., Chehalis, Wash.

North Carolina State Veterinary Medical Association. Goldsboro, N. C. July 7-8, 1936. Dr. J. H. Brown, Secretary, Tarboro, N. C.

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 9-10, 1936. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Veterinary Medical Association of New Jersey. Hotel Monterey, Asbury Park, N. J. July 9-10, 1936. Dr. John G. Hardenbergh, Secretary pro tem., c/o Walker-Gordon Laboratory Co., Plainsboro, N. J.

Virginia State Veterinary Medical Association. Virginia Polytechnic Institute, Blacksburg, Va. July 9-10, 1936. Dr. A. J. Sipos, Secretary, 1102 State Office Bldg., Richmond, Va.

Nebraska Veterinary Medical Association. Annual clinic. Ak-Sar-Ben Field, 63rd and West Center Sts., Omaha, Neb. July 13, 1936. Dr. J. D. Sprague, Secretary, David City, Neb.

San Diego County Veterinary Medical Association. San Diego, Calif. July 14, 1936. Dr. L. K. Knighton, Secretary, 3438 Mountain View Drive, San Diego, Calif.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Fargo, N. Dak. July 14-15, 1936. Dr. Lee M. Roderick, Secretary, North Dakota Agricultural College, State College Station, Fargo, N. Dak.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. July 15, 1936. Dr. L. E. Pike, Secretary, 1220 Bennett Ave., Long Beach, Calif.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 15-16, 1936. Dr. E. A. Caslick, Secretary, Paris, Ky.

Maine Veterinary Medical Association. Sanford, Me. July 16, 1936. Dr. S. W. Stiles, Secretary, Falmouth Foreside, Me.

New York State Veterinary Medical Society. Hotel Lafayette, Buffalo, N. Y. July 16-17, 1936. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Western New York Veterinary Medical Association. Buffalo, N. Y. July 16-17, 1936. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. July 21, 1936. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Veterinary Association of Saskatchewan. University of Saskatchewan, Saskatoon, Sask. July 22, 1936. Dr. Norman Wright, Secretary, 137 20th St. West, Saskatoon, Sask.

Montana Veterinary Medical Association. Kalispell, Mont. July 23-24, 1936. Dr. Hadleigh Marsh, Secretary, Agricultural Experiment Station, Bozeman, Mont.

Western Michigan Veterinary Medical Association. Ionia Fair Grounds, Ionia, Mich. July 30, 1936. Dr. C. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

Poultry Science Association. Virginia Polytechnic Institute, Blacksburg, Va. August 4-7, 1936. Dr. Frank Marcellus, President, Ontario Agricultural College, Guelph, Ontario, Can.

Connecticut Veterinary Medical Association. New London, Conn. August 5, 1936. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

American Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. August 11-14, 1936. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

STATE BOARD EXAMINATION

New Hampshire Board of Veterinary Examiners. State House, Concord, N. H. July 14, 1936. Applications may be obtained from the Secretary. Dr. Fay F. Russell, Secretary, 286 Pleasant St., Concord, N. H.

Dean Francis Dies

Again, just as we are closing the forms of an issue of the JOURNAL, word comes of the death of the dean of one of our veterinary colleges. Dr. Mark Francis, of Texas A. and M. College, died Sunday, June 28, at College Station, Texas.

THE TREATMENT OF CANINE DISTEMPER AND OTHER DISEASES BY THE INTRAVENOUS ADMINISTRATION OF HYDROCHLORIC ACID*

By MILTON M. LEONARD, *Asheville, N. C.*

A survey of the literature of the past several years fails to show any reports, by veterinarians, on the use of hydrochloric acid in distemper. The only references to such work which I could find were by Dr. Burr Ferguson, a physician of Birmingham, Ala., who has used it since 1927 to promote leukocytosis in human infections and who persuaded Dr. K. U. Jones, of the same city, to try it in distemper and a prescription in the handbook of Dr. D. H. Udall, of Cornell University, published in 1912, which reads as follows:

℞

Acid. hydrochlor. dilut.	5.0
Aq. dest.	300.0

Sig.: One teaspoonful to a tablespoonful, every three hours, for a dog with distemper.

The result of Jones' work was a reduction in the death-rate from 75 per cent to 25 per cent in a particularly virulent, septicemic type of the disease.[1]

Dr. Charles DeWitt Colby, of Asheville, was sufficiently impressed by the work of Ferguson in overcoming purulent conditions, apparently through the phagocytic action of the leukocytes stimulated by the acid, to begin the use of this agent in June, 1930. He limited the procedure, at first, to the treatment of cases of human tuberculosis which were complicated by certain suppurative processes. Later, he extended his field to include various allergic conditions and was particularly successful in the cure or amelioration of a large number of cases of asthma and hay-fever.[2] At his suggestion, I began the treatment of dogs suffering from distemper by the intravenous injection of hydrochloric acid in March, 1932. The cases treated during the subsequent years, with the mortality, are enumerated in table I.

These figures speak for themselves with sufficient eloquence, in view of the usual mortality of 50 per cent or over, prior to the use of this agent or of vaccines or sera. The acid, in addition to producing no reaction, has the advantage of very low

*Presented at the seventy-second annual meeting of the American Veterinary Medical Association, Oklahoma City, Okla., August 27-30, 1935.

cost. Doubtless, you are familiar with instances in which the owners of dogs have been willing to pay for one or two doses of serum but, when confronted with the need for a series of doses, have been unable or unwilling to meet the rather considerable expense and have justified themselves with the thought that if the dogs were so sick, they probably would die regardless of treatment.

TABLE I—*Cases of distemper treated with hydrochloric acid, with results.*

YEAR	CASES	RECOVERED	DIED	MORTALITY (%)
1933	98	85	13	13
1934	136	117	19	14
1935 (6 mos.)	48	40	8	17
Totals	282	242	40	14

The solution consists of one part of chemically pure hydrochloric acid to 500 parts of *triple distilled* water. This is three times the strength of the solution used in human beings. The usual dose is 10 cc and I have found that this dosage is well tolerated, regardless of the age, size or breed of the dog. I have given 20 cc in large dogs or in those whose symptoms were severe. The solution is injected slowly into any vein, using the usual technic for intravenous medication. Care should be taken to see that none of the solution is injected outside the vein, as slight temporary discomfort will be noted if this happens. Examination of the blood confirmed the observations made on man, that a leukocytosis is produced with a marked increase in both polymorphonuclear cells and lymphocytes. Blood counts made when the animals were first seen, according to the scheme of Schilling, all showed the presence of juvenile and "stab" cells, which gave way to the types mentioned above with the establishment of convalescence. A decrease in the number of monocytes also is seen. No blood chemistry was done, so that I know nothing of the possible effect on the sugar content. Typical findings are presented in table II.

Colby demonstrated phagocytosis in the bacterial conditions encountered, confirming the observations of Ferguson. Of course, with a virus or ultramicroscopic organism, such action can only be assumed. When mixed infection occurs, however, as it does in the advanced stages of distemper, phagocytosis may be a

potent factor in recovery. Another element, however, is suggested by Colby, which may explain what takes place in the early stage, prior to bacterial invasion. He referred to the work of Victor Vaughn,[3] who said:

> When a foreign protein permeates a body cell, that cell is stimulated to produce a ferment with which to destroy that foreign protein.

Colby says:

> There is no ferment action without a catalyst and in the body the catalyst is always an acid. Hydrochloric acid is the only stable mineral acid produced by the body. It is the catalyst for pepsogenic action on gastric contents. Can it possibly be the catalyst for other body ferments?

TABLE II—*Blood findings in two typical cases.*

CASE	DATE	BASOPHILES	EOSINOPHILES	NEUTROPHILES				LYMPHOCYTES	MONOCYTES	NUCLEAR INDEX
				MYELOCYTES	JUVENILES	"STABS"	SEGMENT			
402	7– 4–35	0	0	0	7	38	17	14	24	0.31
	7– 8–35	0	2	0	0	9	46	34	9	5.1
498	8– 5–35	0	0	0	5	10	66	9	10	6.6
	8–15–35	0	0	0	0	5	70	21	2	14.0

Turning from theoretical discussion, let us direct our attention to the practical consideration of the facts, as presented by the cases themselves. I shall refer very briefly to the following cases:

CASE 1—BOSTON TERRIER

July 5—No appetite, ocular discharge, slight nasal discharge, weakness of hind quarters. Temperature 103.8° F. Given 10 cc hydrochloric acid solution.

July 8—Appetite better, general condition better, discharge less. Temperature 102.4° F. Given 10 cc hydrochloric acid solution.

July 11—Appetite good, still better except for weakness of hind legs, but getting stronger. Temperature 101.0° F. Given 10 cc hydrochloric acid solution.

July 16—Apparently normal, legs almost normal strength. Temperature 100.2° F.

CASE 2—GERMAN SHEPHERD (6 MONTHS)

June 13—Sluggish, appetite poor, discharge from eyes and nose, bowels slightly loose. Temperature 103.2° F. Given 10 cc hydrochloric acid solution.

June 14—Appetite better, livelier, slight discharge from eyes and nose, bowels slightly loose. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
June 15—Appetite fairly good, slight discharge from eyes, less from nose, bowels normal.
June 16—Appetite fairly good, livelier, slight discharge from eyes, practically none from nose, bowels normal. Temperature 101.6° F. Given 10 cc hydrochloric acid solution.
June 17—Appetite almost normal, slight discharge from eyes, nasal discharge ceased.
June 18—Appetite normal, ocular discharge almost ceased, lively. Temperature 101.2° F. Given 10 cc hydrochloric acid solution.
June 22—Apparently normal. Temperature 100.4° F.

CASE 3—GERMAN SHEPHERD

Jan. 31—Having fits, slight discharge from eyes and nose. Temperature 106.0° F. Given 15 cc hydrochloric acid solution.
Feb. 1—Only two fits, better. Temperature 103.8° F. Given 10 cc hydrochloric acid solution.
Feb. 2—No fits, appetite fair. Temperature 102.8° F. Given 10 cc hydrochloric acid solution.
Feb. 3—Appetite good, becoming lively. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
Feb. 4—Appetite normal, slight discharge. Temperature 102.4° F. Given 10 cc hydrochloric acid solution.
Feb. 5—Condition same. Temperature 102.6° F.
Feb. 6—Better, discharge ceased. Temperature 102.0° F.
Feb. 7—Apparently normal, except for loss of weight. Temperature 101.0° F.

CASE 4—DANE (MALE, 1 YEAR)

June 14—Appetite poor, thin, listless, slight nasal and ocular discharges, bowels normal. Temperature 103.0° F. Given 10 cc hydrochloric acid solution.
June 16—Appetite better, livelier, less discharge, otherwise little change. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
June 18—Appetite fair, lively, bowels slightly loose. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
June 20—Appetite poor, listless, bowels loose, more discharge. Temperature 101.8° F. Given 10 cc hydrochloric acid solution.
June 22—Appetite better, livelier, bowels better. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
June 24—Worse. Temperature 104.6° F. Given 10 cc hydrochloric acid solution.
June 25—Better. Temperature 103.6° F. Given 10 cc hydrochloric acid solution.
June 27—Better. Temperature 101.8° F. Given 10 cc hydrochloric acid solution.
June 29—Appetite normal, bowels normal, discharges ceased, lively. Temperature 100.6° F.

CASE 5—LARGE GERMAN SHEPHERD

Jan. 25—Having fits for several days, at other times listless, no appetite, nasal and ocular discharges. Temperature 105.6° F. Given 20 cc hydrochloric acid solution.
Jan. 26—Fewer fits, livelier, but no change otherwise. Temperature 102.6° F. Given 10 cc hydrochloric acid solution.
Jan. 27—Only one fit, beginning to eat. Temperature 102.8° F. Given 10 cc hydrochloric acid solution.

Jan. 28—No fits, ate better, nasal and ocular discharges less, livelier. Temperature 101.6° F. Given 10 cc hydrochloric acid solution.
Jan. 29—Appetite almost normal, lively, discharges less. Temperature 101.0° F. Given 10 cc hydrochloric acid solution.
Jan. 30—Condition unchanged. Temperature 101.6° F. Given 10 cc hydrochloric acid solution.
Jan. 31—Appetite normal, otherwise same. Temperature 101.4° F.
Feb. 2—Appetite normal, not quite so playful, otherwise same. Temperature 103.0° F. Given 10 cc hydrochloric acid solution.
Feb. 3—General condition slightly worse. Temperature 104.0° F. Given 10 cc hydrochloric acid solution.
Feb. 4—Slightly worse. Temperature 105.0° F. Given 20 cc hydrochloric acid solution.
Feb. 5—Much better. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
Feb. 6—Better. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
Feb. 7—Better. Temperature 102.0° F. Given 10 cc hydrochloric acid solution.
Feb. 8—Appetite normal, discharges clearing up. Temperature 101.4° F. Given 10 cc hydrochloric acid solution.
Feb. 9—Discharges about ceased. Temperature 101.0° F. Given 10 cc hydrochloric acid solution.
Feb. 10—Apparently normal. Temperature 100.6° F.

A rather striking case was that of a six-week-old, cross-bred pup which was admitted with its mother, the latter being in an advanced stage of the disease. Both animals were given the usual doses of hydrochloric acid by the usual technic, with the result that the mother recovered and the puppy remained well. Of course, the matter of natural immunity should be considered here, and I should have a larger number of instances of such exposure before drawing any conclusions as to the value of hydrochloric acid as a prophylactic or immunizing agency.

These case reports cover only cases of distemper which have been treated at the hospital. The results in the cases which have been presented for diagnosis and treatment and which have been taken out immediately after medication have been equally as satisfactory as far as I have been able to ascertain by inquiry. Many have never received more than three doses and have made complete recoveries. I have never observed chorea as a sequal of distemper in any case treated with hydrochloric acid.

Other conditions in which this treatment has been of decided benefit are jaundice; eczema; chorea; running fits, which I believe to be due to distemper with secondary infection; paralysis, with incoördination of the hind quarters and no other symptoms except the inability to stand; cases of purulent preputial discharge, with evident lowering of systemic resistance; preoperative preparation and post-operative care, with more rapid healing and the earlier removal of stitches. In eczema and

chorea the same factors may be operative as in human allergy. The results with preputial discharge were particularly impressive, as cases which were resistant to various forms of local treatment cleared up promptly with an average of about three injections of hydrochloric acid.

I do not wish to appear, in advocating this treatment, as holding it to be superior to the homologous serum which I believe to be specific, but I think that the ideal method is a combination of the two, which, to my mind, would prove to be better than either one alone.

SUMMARY

In this paper are reported:

1. A group of 282 cases of canine distemper, with 242 complete recoveries and 40 deaths, or a mortality of 14 per cent, following the intravenous administration of hydrochloric acid.
2. The use of hydrochloric acid in several cases of jaundice, eczema, chorea, paralysis, running fits and perpetual discharge.
3. The low cost of such treatment compared to the use of serum.

ACKNOWLEDGMENT

At this time, I wish to acknowledge my indebtedness to the personnel of the William LeRoy Dunn Memorial Clinic, of Asheville, N. C., for suggestions, advice and technical laboratory assistance, in my work, with hydrochloric acid.

REFERENCES

[1]Jones, K. U.: Med. Mentor, 1 (1930).
[2]Colby, C. D.: Treatment of asthma and kindred conditions by intravenous administration of hydrochloric acid. South. Med. & Surg., xciv (1932).
[3]Vaughn, V.: Split Protein Products in Immunity and Disease.

Iowa Veterinarian Wins Trapshooting Tournament

Dr. John Patterson, of Des Moines, Iowa, for the second time in three years, won the Iowa State 16-yard trapshooting championship at the Des Moines Gun Club grounds, on June 21, by crashing 75 straight clay birds in a shootoff of a six-way tie at 194 in the regular 200-target card. It was a wonderful exhibition of nerveless shooting upon the part of the well-known Iowa veterinarian, according to competent witnesses. Dr. Patterson won the crown in 1934, his first try at the title. His performance this year was all the more remarkable, as a stiff, chill wind blew most of the day, targets were erratic in their flight, and burned powder was blown back in the faces of the shooters. Congratulations, John.

THE CELLULAR ELEMENTS AND HEMOGLOBIN IN THE BLOOD OF CHICKENS WITH SPONTANEOUS TUBERCULOSIS*

By CARL OLSON, JR., *and* WILLIAM H. FELDMAN
The Mayo Foundation, Rochester, Minn.

The morphologic pathology of spontaneous tuberculosis of chickens is fairly well understood in so far as the gross and microscopic character of the morbid changes are concerned. There are also considerable data on the anatomic distribution of the lesions in the respective parenchymatous organs. However, there exists a scarcity of authentic information pertaining to the alterations that may ensue in the cellular elements and hemoglobin of the circulating blood as a consequence of a spontaneous tuberculous infection. Since the spontaneous form of the disease in the chicken is usually insidious in its inception and is characterized by its chronicity and protracted course, it would seem of interest to know something of the effect of the infection on the hematopoietic tissues and the circulating blood.

For the purpose of obtaining additional information concerning the total number of cells and the value for hemoglobin in the blood of chickens affected with spontaneous tuberculosis the studies reported in this paper were made.

REVIEW OF THE LITERATURE

The available literature contains only meager reports concerning the number of cells and amount of hemoglobin in the blood of chickens affected with spontaneous tuberculosis. Only slightly more work has been done in this regard with the experimentally produced disease.

Moore[1] observed that in advanced cases of tuberculosis an anemia was manifest, the erythrocytes being reduced in number to 1,100,000 per cubic millimeter of blood. He also observed a slight leukocytosis, especially of the polynuclear forms. The values for hemoglobin ranged from 35 to 70 per cent, as measured by Gower's test.

Ellermann and Bang[2] also mention that leukocytosis may be associated with the disease in some cases. Hirschfeld and Jacoby[3] found leukocytosis in experimentally infected chickens, leukocytes numbering up to 75,000 per cmm of blood. They mention also that approximately half of the leukocytes consisted of lymphocytes and large mononuclear cells, and that the number of mast cells was unusually great.

*Received for publication, August 15, 1935.

Burckhardt[4] studied 23 chickens with experimentally produced tuberculosis and one with spontaneous tuberculosis. He found a marked leukocytosis, usually to the extent of 100,000 to 200,000 leukocytes per cmm of blood. In one instance he observed a value of 367,000 leukocytes per cmm. The pseudo-eosinophils were greatly increased. The lymphoid cells (including lymphocytes and monocytes) also were increased. He considered some of these lymphoid cells to be the predecessors of erythrocytes and that the blood picture of fowl tuberculosis was a mixture of leukocytosis and "pernicious anemia." His opinion of the relationship between the tuberculosis and leukosis of the chicken was proved unfounded by the later work of Hirschfeld and Jacoby. An interesting feature was reported by Burckhardt in his experimental studies in that, in chickens intravenously inoculated, he observed, in the second week following inoculation, a drop in the number of leukocytes, and a rise to high levels in the third and fourth weeks. In some instances there was a drop in the number of leukocytes just preceding death.

Römer[5] described the changes in the blood in chronic tuberculosis as consisting of a marked increase in the number of large lymphocytes and pseudo-eosinophils at the expense of the small lymphocytes. The eosinophils, the basophils, and the erythrocytes he found to be essentially normal in number.

Yakimoff and Rastégaïeff[6] reported their findings in one case of spontaneous avian tuberculosis. They observed a great increase in the number of cells which they designated as monocytes. They gave the following values for the differential count: lymphocytes, 4.2 per cent; monocytes, 56.9 per cent; polynuclear cells, 33.4 per cent; eosinophils, 0, and mast cells, 0.4 per cent.

Animals Studied

The animals used in this study consisted of eight adult female chickens, of the Rhode Island Red breed, between two and three years of age. They were secured from a farm flock in which the incidence of avian tuberculosis was relatively high. A few days before the birds were purchased, they had been injected intradermally with avian tuberculin* and each had reacted positively. A total of ten birds that had reacted positively to the test were obtained, but two died with extensive lesions of tuberculosis before the studies of the blood began. The eight surviving

*The tuberculin test was administered by Dr. S. H. Burgess, of the Bureau of Animal Industry, U. S. Department of Agriculture. The tuberculin used was that furnished by the U. S. Department of Agriculture for the routine testing of chickens for tuberculosis.

chickens, with the exception of one, were in fairly good condition and exhibited few if any objective symptoms of disease.

As soon as the chickens were brought to the laboratory, each was placed in a separate cage and kept there for a period of two weeks before the experimental observations were started.

Methods and Hematologic Examination

The methods of enumerating the various elements of the blood were those found to be satisfactory in previous studies.[7, 8]

The determination of the number of leukocytes per cmm of blood was made by the use of the phloxine diluting solution. This solution was composed of 50 mg of phloxine, 5 cc of formalin (40 per cent solution) and 95 cc of Ringer's solution. The ordinary erythrocyte counting-pipets were used in which the samples of blood obtained from the wing vein were diluted 200 times. The filled pipets were allowed to remain at ice-box temperature overnight before counting to obtain better staining of the cells. This solution stains the acidophilic granulocytes of the blood in such a manner that they may be recognized in the counting chamber. Their number in 0.9 cmm of the counting-chamber was noted. By use of the number thus obtained and the percentage value of the cells found by the differential count of leukocytes, the total number of leukocytes per cmm of blood was calculated.

The number of thrombocytes was estimated by observing the number of these cells occurring per 100 leukocytes in the blood-smear preparation. With the ratio found, the number of the thrombocytes per cmm of blood was calculated from the value of leukocytes per cmm of blood.

The erythrocytes were counted in the usual manner in the counting-chamber. The same pipet preparation that was used in the counting of acidophilic granulocytes was used in the enumeration of erythrocytes.

The differential counts were made from blood smears stained with the May-Grünwald and Giemsa solution.[9] One hundred cells were counted. A description of the leukocytes in the blood of the chicken is given in a previous report.[8].

The measurements of hemoglobin were made with the Sheard and Sanford photo-electric hemoglobinometer. This instrument was demonstrated by previous work[7] to be applicable to the measurement of the hemoglobin in chickens' blood.

Samples of blood were taken on each of four successive days, and these were obtained at about the same hour on each day. Food and water were available to the birds at all times, so that feeding or fasting periods would not affect the blood-cell counts.

Blood Cultures

Approximately 2 cc of blood was obtained from one of the wing veins of each chicken for the purpose of attempting cultures for the isolation of *Mycobacterium tuberculosis*. In chickens infected experimentally with *M. tuberculosis* we had previously succeeded in recovering the organism from the peripheral bloodstream by inoculating the whole blood on Herrold's egg-yolk agar media to which had been added 3 per cent glycerin. Since the blood was secured aseptically, treatment with chemical reagents to eliminate contaminating bacteria was not necessary. Generally speaking, the same procedure was followed in attempting to recover *M. tuberculosis* from the peripheral blood of the eight chickens which were studied hematologically. Each specimen of blood was drawn into a syringe containing a sufficient amount of potassium oxalate to prevent coagulation and was then used to inoculate slants of Herrold's medium. Approximately 0.5 cc of blood was permitted to flow over the surface of each tube of media. After sufficient drying had occurred, the respective tubes were capped and incubated at 38° C. for a period of eight weeks. Blood cultures were attempted for each of the eight birds on four successive days. In only one instance was *M. tuberculosis* obtained. This occurred from a single day's specimen of the blood of chicken 2. One colony of acid-fast bacilli occurred on each of the four slants of media. Subcultures of these organisms proved pathogenic for rabbits and chickens but not for guinea pigs.

Pathologic Anatomy

As soon as the hematologic examinations were complete, each chicken was killed, the feathers were removed, and a careful postmortem examination was made. Lesions were noted and tissues were preserved from all organs for subsequent histologic study. From each tissue preserved duplicate sections were made, one being stained with hematoxylin and eosin and the other with a carbolfuchsin-hematoxylin combination to reveal the presence of any acid-fast bacteria that might be present.

The distribution of lesions of tuberculosis in the respective chickens, as determined by the gross findings at necropsy and by microscopic examination of the various tissues, is summarized in table I. Tuberculous lesions containing characteristic bacillary forms were present in all of the chickens except one (5). The disease in two chickens (1 and 6) was limited to the lungs, whereas the lesions in the remaining chickens were of an aggravated character and were extensively distributed. In only three of the chickens were there tuberculous foci in the bone-

TABLE I—*Summary of findings at necropsy and the results of cultures of the blood of eight adult chickens, each of which had reacted positively to a diagnostic dose of avian tuberculin administered intradermally.*

CHICKEN	PHYSICAL APPEARANCE	SITUATION AND TYPE OF TUBERCULOUS LESIONS	BLOOD CULTURES	EGG PRODUCTION
1	Good	Nodular* lesions in lungs	Negative	Limited
2	Poor	Extensive and progressive lesions in lungs, spleen, liver and intestines	Positive once	
3	Good	Nodular lesions in spleen and intestine; miliary foci in lungs	Negative	
4	Poor	Widespread generalized infection	Negative	
5	Fair	No lesions demonstrable	Negative	Limited
6	Good	Disease limited to one nodule in one lung	Negative	
7	Good	Nodular lesions in intestine; miliary lesions in liver and spleen	Negative	
8	Fair	Extensive lesions in liver, spleen and intestine; few foci in lungs	Negative	Limited

*The word "nodular" is used to designate lesions of tuberculosis of greater dimensions than is ordinarily meant by the term "miliary." Generally speaking, a nodular lesion represents a tuberculous focus measuring more than 0.2 cm in diameter.

marrow, as determined by an examination of the myeloid tissue of the femur. Egg production of a limited character was noted in only two of the chickens that were affected with tuberculosis.

RESULTS OF HEMATOLOGIC OBSERVATIONS

The results of the four daily counts on each of the birds were averaged and the average values of the various elements of the blood of the different birds are presented in table II. In no instance was the value of a daily reading of any of the elements of the blood more divergent from the average value than could be explained by means of the coefficient of variation of the method applied.*

The results of the hematologic observations were not the same in each of the individual fowls. The erythrocyte counts and values for hemoglobin were within the limits of those noted in normal chickens. The thrombocyte counts of the tuberculous fowls were likewise within normal limits. The thrombocyte counts of three of the chickens (3, 7 and 8) may represent high normal values and have no special significance.

The total and differential leukocyte counts revealed data of

*The coefficient of variation for each of the various determinations is presented in other reports (Olson[7, 8]).

TABLE II — *Results of four daily examinations of the blood of eight female chickens reacting positively to the tuberculin test.*

CHICKEN	ERYTHROCYTES*	HEMOGLOBIN†	THROMBOCYTES‡	LEUKOCYTES‡	DIFFERENTIAL COUNT (IN PER CENT UNLESS OTHERWISE NOTED)					EXTENT OF TUBERCULOSIS
					LYMPHOCYTES	HETEROPHILS	EOSINOPHILS	BASOPHILS	MONOCYTES	
1	2.37	8.57	30,800	40,700	46.8 *19,047*	31.7 *12,901*	2.0 *814*	6.5 *2,645*	13.0 *5,291*	Slight
2	2.89	8.97	36,000	92,700	31.6 *29,293*	37.0 *34,299*	.2 *185*	1.0 *927*	30.2 *27,995*	Extensive
3	2.56	9.62	42,300	31,700	45.2 *14,328*	35.7 *11,316*	3.2 *1,014*	1.7 *538*	14.2 *4,500*	Extensive
4	2.65	9.62	29,000	64,700	38.6 *24,974*	38.0 *24,586*	2.2 *1,423*	1.5 *970*	19.7 *12,745*	Extensive
5	3.01	10.61	33,500	31,000	62.9 *19,499*	22.2 *6,882*	2.2 *682*	5.0 *1,550*	7.7 *2,387*	Negative
6	2.77	10.77	29,000	39,000	41.3 *16,107*	36.4 *14,196*	1.2 *468*	3.1 *1,209*	18.0 *7,020*	Slight
7	2.84	9.36	41,800	42,900	51.0 *21,879*	28.1 *12,054*	3.2 *1,372*	2.2 *943*	15.5 *6,649*	Moderate
8	3.11	10.62	54,100	65,800	38.1 *25,069*	36.3 *23,885*	2.3 *1,513*	.7 *460*	22.6 *14,870*	Extensive
Normal values, adult female chicken	Mean 2.81§	9.11§	26,500	19,800	64.6	22.8	1.8	1.7	8.9	
	S. D.‡ ±.02	±.17	±300	±300	±.4	±.3	±.1	±.1	±.1	
	.30	1.14	7,900	9,600	8.6	7.9	1.6	1.2	3.5	

Italicised numbers express total values of respective leukocytes per cubic millimeter of blood.
*Millions per cmm of blood.
†Grams per 100 cc of blood.
‡Per cubic millimeter of blood.
§Normal value for the period of year during which the observations were made on the tuberculous chickens.
‡ S. D. Standard deviation of normal mean values.

much interest. Chicken 5, which reacted to the tuberculin test but in which no evidence of tuberculosis could be found at necropsy, was essentially normal as regards the hematologic findings. In two chickens (1 and 6) in which tuberculosis was only slight in extent, the total leukocyte count was higher than normal. There was an increase in the percentage of heterophils and monocytes and a decrease in the percentage of lymphocytes. Although the relative number of lymphocytes was less than normal, the total number was slightly more than the normal value given. The percentage of basophils was rather markedly high for chicken 1 and only slightly less so for chicken 6. The relative values for eosinophils in these two birds were essentially normal, although the total values may be slightly higher than the value given as normal.

Four of the chickens (2, 3, 4 and 8) were found at the time of necropsy to have extensive, widespread tuberculous infection. Three of these (2, 4 and 8) showed marked leukocytosis. The chief increase was in the heterophils and monocytes, although the absolute values for the other types of leukocytes were also higher than normal. One bird (3) showed a mild leukocytosis, the increase in the number of cells being in the heterophils and monocytes; the total number of lymphocytes was essentially normal.

No abnormal cell forms were observed in the blood of the chickens studied. Slight variations in the morphologic appearance of the various leukocytes were observed, but these were also noted in previous work with the blood of normal chickens.

COMMENT

The most striking finding on examination of the blood in avian tuberculosis is the leukocytosis, in which the values for monocytes and heterophils are increased. The leukocytosis was correlated roughly in a direct ratio to the extent of the disease. For example, in one chicken (2) that had extensive tuberculosis the increase in the number of these cells was most marked, whereas in another (1), which had only slight tuberculosis, the increase was not so striking. Certain exceptions do occur, since one chicken (3), with extensive tuberculosis, had only a mild leukocytosis.

The finding of leukocytosis in avian tuberculosis confirms the observations of some of the previous investigators. Hirschfeld and Jacoby considered the leukocytosis to be mainly in respect to lymphocytes and mononuclear cells. The cells designated by Römer as large lymphocytes might well be similar to what we have considered as monocytes.

Anemia was not present in any of the tuberculous fowls studied. In some of these fowls the disease was well established and probably was of long duration. The fact that it is reported by Moore as occurring in some cases, coupled with our failure to observe it, leads us to believe that it is a rather rare occurrence.

The hematologic findings in regard to chicken 5 are of interest in that they may be considered as essentially normal. It will be recalled that necropsy of this fowl failed to reveal any evidence of tuberculous infection although a careful search was made. The positive reaction to tuberculin in this case may have resulted as a consequence of a very recent infection or because of the presence of obscure lesions that escaped detection. However, the explanation of such a reaction is not entirely satisfactory. That many chickens without demonstrable lesions of tuberculosis may react in a characteristic manner to the tuberculin test was reported previously by one of us (Feldman[10]).

SUMMARY

Four daily observations were made on the numbers of erythrocytes, thrombocytes and leukocytes, and on the amount of hemoglobin in the blood of eight female chickens which reacted positively to intradermal injections of tuberculin. Blood cultures of bacilli of avian tuberculosis were made at the time the samples of blood were taken. The chickens were subsequently killed and the extent of tuberculosis was noted at necropsy. The hematologic findings were then compared to those for normal chickens and were correlated with the extent of the tuberculous process.

One chicken failed to give macroscopic or microscopic evidence of tuberculosis at necropsy and also presented essentially normal hematologic findings.

Those chickens with tuberculosis revealed leukocytosis, especially in regard to the heterophil granulocytes and monocytes. The degree of leukocytosis was roughly in a direct ratio with the extent of the tuberculosis.

Anemia was not manifested by any of the tuberculous fowls studied.

Attempts to obtain, by culture, bacilli of avian tuberculosis from the circulatory blood failed except in one instance.

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World's Poultry Congress

The United States will be represented at the Sixth World's Poultry Congress, Leipzig, Germany, July 24 to August 2, 1936, by nine well-known scientists from different parts of the country. They are:

Dr. J. R. Beach, Department of Veterinary Science, University of California, Berkeley, Calif.

Dr. G. C. Buckner, Kentucky Agricultural Experiment Station, Lexington, Ky.

Mr. John A. Hannah, Michigan State College, East Lansing, Mich.

Dr. Thomas W. Heitz, Bureau of Agricultural Economics, U. S. Department of Agriculture, Washington, D. C.

Dr. Hugh C. McPhee, Chief, Animal Husbandry Division, U. S. Bureau of Animal Industry, Washington, D. C.

Dr. J. R. Mohler, Chief, U. S. Bureau of Animal Industry, Washington, D. C.

Prof. James E. Rice, Professor Emeritus, Cornell University. (Trumansburg, N. Y.)

Prof. R. B. Thompson, Oklahoma College of Agriculture, Stillwater, Okla.

Dr. C. W. Warburton, Director of Extension, U. S. Department of Agriculture, Washington, D. C.

Previous congresses have been held at The Hague, Holland; Barcelona, Spain; Ottawa, Canada; London, England, and Rome, Italy. There is considerable sentiment in favor of holding the 1939 congress in the United States.

Calling Doctor Recht

If this notice should come to the attention of Dr. Henry R. Recht, Jr., of Cedar Grove, N. J., please communicate with your father at once. If any reader of the JOURNAL knows of the present whereabouts of Dr. Recht, kindly communicate with the Secretary of the A. V. M. A.

STUDIES OF PULLORUM DISEASE

II. The Incidence of Salmonella Pullorum in Eggs from Infected Hens*

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Although numerous papers have appeared on the incidence of *Salmonella pullorum* in the egg of the reacting hen, the true picture has not been obtained, due largely to an adequate technic. A further study of this problem appeared worthwhile when a new technic for the isolation of *S. pullorum* was developed by Mallmann, Thorp and Semmes,[1] whereby the obscuring of positive tests by contaminating organisms could be avoided. Further, it was believed that a study of pullorum disease in the egg during the hatching period would be of interest and the results obtained might be of value in understanding the spread of pullorum disease.

EXPERIMENTAL

The hens selected for this study were reactors from a Michigan flock. All of the reactors were carefully checked, using both the rapid whole-blood test and the slow test or test-tube method. The reacting flock consisted of 42 S. C. Rhode Island Reds. During the early part of 1934, they contracted infectious bronchitis (infectious laryngotracheitis), which was detrimental to their production and to the hatchability of their eggs. They were fed a suitable diet which, in the remainder of the flock, gave a satisfactory production of eggs with good hatchability. All eggs from the reactor flock were saved and settings were made every two weeks. The eggs were set in a cabinet-type machine for 18 days, when they were transferred to a sectional-type incubator (still air) for hatching. The infertile eggs were candled out on the tenth day of incubation.

The method of culturing the eggs differed considerably from that recommended by Rettger,[2] Hadley and Caldwell,[3] and Runnells and Van Roekel.[4] Their methods consisted essentially in disinfecting the exterior of the egg and then puncturing the end with a sterile probe and opening with sterile forceps. This method, due to the amount of manipulation, may allow considerable

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contamination to enter the egg. To avoid this the following procedure was used:

All eggs were disinfected by submerging them in a 1:1,000 mercuric chloride solution for ten to 30 minutes. The eggs were lifted aseptically from the mercuric chloride solution to small beakers of such size that the beaker served as an egg-cup for culturing. The eggs protruded above the beaker to the extent of about two-thirds their height. This prevented the lips of the beaker from coming in contact with the shell at the point of culturing. The egg was placed in the beaker with the air-sac uppermost. While the eggs were still moist with the mercuric chloride solution, a sharp-pointed gas flame was applied to the shell covering the air-sac. The heat of the flame caused a rapid expansion of the air within the air sac, with the result that the shell was forcibly removed over the air-sac with a report like a firecracker. Thus the shell was removed rapidly and effectively without any danger of contamination. Very frequently the inner membrane was ruptured so the egg was ready for culturing without further preparation.

Several methods of examination were used to determine which gave the most satisfactory results. The procedures were as follows:

Direct method: 1—A loopful of yolk was smeared on the surface of a brilliant-green, liver-infusion agar, prepared as recommended by Mallmann, Thorp and Semmes.[1] The typical or atypical colonies which appeared, after incubation for 48 hours at 37° C., were transferred to lactose broth fermentation tubes containing Andrade's indicator. If they failed to ferment this sugar with the production of acid or acid and gas, they were transferred to maltose, sucrose, mannite and dextrose broth for further identification.

Indirect method: 2—Brilliant-green, beef-extract broth enrichment procedure. Approximately 1 cc of yolk was transferred to about 10 cc of brilliant-green, beef-extract broth prepared according to Mallmann.[5] After incubation for 48 to 72 hours at 37° C., brilliant-green, liver-infusion agar plates were smeared to obtain the *S. pullorum* in pure culture. All colonies were cultured as previously described.

3—Dextrose-broth enrichment procedure. After the removal of the 1-cc portion of egg yolk for the brilliant-green, beef-extract broth, the remaining yolk and egg white were poured into large-mouthed Erlenmyer flasks containing 100 cc of dextrose, beef-extract broth. After incubation for 48 hours at 37° C., bril-

liant-green, liver-infusion agar plates were smeared and *S. pullorum* was isolated and identified as before. Again after incubation of the dextrose broth cultures for seven days at 37° C., another set of brilliant-green, liver-infusion plates was made as before.

In the data presented, the results on infertile and fresh eggs are the total number of positives obtained by all the procedures listed. In all cases, all of the listed procedures were applied to all infertile and fresh eggs tested. The dead germs in the case of eggs where the embryos died at an early stage were handled in the same manner.

In the examination of the chicks for the presence of *S. pullorum*, all chicks were killed as soon as they were hatched to avoid any spread of the disease from the infected to the clean chicks while they were in the incubator. The liver, heart and retained yolk were cultured on brilliant-green, liver-infusion agar plates. All suspected colonies were checked on the sugars previously listed to establish their identity. The chicks that died in the shell were examined in like manner. The brilliant green in the culture medium acts selectively for *S. pullorum* and prevents overgrowths by contaminating organisms. In no instance, either in infertile eggs or mature chicks, was evidence of any other organisms found, although other organisms may have been present in the eggs and chicks at the time of examination.

With the exception of 136 eggs that were examined fresh, all eggs were incubated for hatching. At the end of ten days, the infertile eggs were removed from the incubator and examined, and at the end of 18 days, the dead germs were removed and examined. All eggs that failed to hatch were cultured at the end of the incubation period.

In the tables presented, the data are assembled into three divisions, namely, infertile eggs, dead germs and dead in shell, and freshly hatched chicks.

Presentation of Data

The percentage of infection in the egg has been studied extensively and the results obtained are extremely variable. This variability is due largely to two factors: (1) extent and severity of the disease in the flock studied, and (2) the technic used in making the examination. In several papers a satisfactory method of isolation has been used, but unfortunately the resulting figures were interpreted incorrectly. Runnells and Van Roekel,[4, 6] in their studies, followed the same procedure of isolation in all

their work, but used varying periods of incubation of the eggs prior to examination. Their method of examination consisted briefly in culturing the entire egg in 30 to 40 cc of nutrient broth, then smearing plates for isolation of the organism at the end of 48 hours, and seven days of incubation. The influence of incubation on the incidence of the organism in the egg is vividly demonstrated in table I.

TABLE I—*Influence of incubation on incidence of Salmonella pullorum in the egg (Runnells and Van Roekel).*

PERIOD OF INCUBATION OF EGGS BEFORE TESTING	169 EGGS FROM REACTING RHODE ISLAND RED HENS (% POSITIVE)	305 EGGS FROM REACTING WHITE LEGHORN HENS (% POSITIVE)
Fresh	20.9	20.1
7 days	19.4	7.9
14 days	41.0	18.7
21 days	49.0	19.7
Averages	33.7	14.0

Evidently Runnells and Van Roekel took an average of the four different methods as representing the degree of infection rather than selecting the results from the best procedure. Their final data on the infection in each flock of hens measure not the extent of the infection, but the average efficiency of the various periods of incubation in promoting growth of the organism prior to examination. Apparently, in the case of the incubated eggs, the data represent only those eggs removed at the different periods during the period of incubation. The seven-day incubation appears to represent infertiles largely, the 14-day observations dead germs, and the 21-day the chicks dead in the shell. The averages do not include those eggs in which chicks were produced. Seemingly their figures do not show that the incidence of the disease is higher in the R. I. Red hens, but on the contrary they show a similar amount of infection if a comparable method of examination is used. The only data that can be compared are those of the fresh eggs. These data show 20.9 per cent positive for the R. I. Red hens and 20.1 per cent positive for the White Leghorn hens.

In a study by Tittsler, Heywang and Charles,[7] 929 eggs were cultured by direct transfer to agar slants after the eggs were incubated ten days, 440 eggs were examined by transferring the whole yolk to 25 cc of broth and culturing after suitable in-

cubation, and 191 eggs were examined after incubation for hatching. The results of each method were as follows:

1. Direct transfer.....3.6 per cent positive
2. Whole yolk ..... 6.7 per cent positive
3. Hatching .....10.5 per cent positive
   Average ..... 5.2 per cent positive

They conclude that the extent of the infection was 5.2 per cent; whereas the figure 5.2 represents the average efficiency of the three methods used.

In a study by Rettger,[2] several procedures were tried. In an examination of fresh eggs, the entire yolk was tested by placing it aseptically into nutrient broth and incubating for a suitable period prior to the isolation of the organism on smear cultures. By this procedure, of 3,510 eggs examined, 109 were positive, a percentage of 9.5. In a second procedure the eggs were cultured by direct smear of the yolk on agar, but they were incubated for varying periods prior to examination. The results of the latter procedure are shown in table II.

TABLE II—*Influence of incubation on incidence of Salmonella pullorum in the egg (Rettger).*

PERIOD OF INCUBATION	EGGS TESTED	POSITIVE	
		No.	%
1 week	1746	48	2.8
2 weeks	2166	28	1.3
3 weeks	1984	71	3.6

The differences in percentage infection, as revealed by longer incubation periods, were inconsequential.

Weaver and Weldin[8] studied 20 reactor hens from mixed flocks and found 53 infected eggs in 775 eggs examined, an infection of 6.8 per cent. Their technic of isolation was not described. Kaupp and Dearstyne,[9] in a comparative study of Single Comb Rhode Island Reds and Single Comb White Leghorns, found 7.31 per cent of 1,313 eggs of the former breed infected, and 5.23 per cent of 2,505 eggs of the latter breed. The average for the two breeds was 5.94 per cent positive. Their technic of isolation was not stated. Gwatkin,[10] in an examination of 420 eggs from reacting hens, found 4.76 per cent infected. His technic consisted briefly in incubating the yolk in 20 cc of nutrient broth for 48 to 72 hours and then smearing on agar plates or agar slants.

Because of the marked variability in the results obtained by the various workers and the various procedures used, a comparative study of methods was made, collecting material from the same egg for each test. Brilliant-green, liver-infusion media were used. It was demonstrated by Mallmann, Thorp and Semmes[1] that brilliant green in proper concentration does not inhibit *S. pullorum*, but does inhibit *Escherichia coli* and all Gram-positive contaminants that are commonly encountered in examination of material of this nature, particularly in culturing the organs of chicks or culturing from incubated broth to which whole yolk has been added. The use of liver infusion in the medium offers a much more favorable pabulum for the growth of *S. pullorum*. It has been the experience of the senior author that frequently *S. pullorum* could be obtained from liver-infusion, smear plates when beef-extract or beef-infusion, agar plates were negative. It is quite conceivable that *S. pullorum* in the egg, particularly the infertile egg, is under adverse conditions, whereby the lag period of growth has been lengthened markedly and unless very favorable growing conditions are used, the organism dies or its lag period of growth is longer than the period of incubation used, with the result that the culture is discarded before growth appears. In the light of the data to be presented, it is certain that the low incidences obtained by Gwatkin, Kaupp and Dearstyne, Rettger, and others were largely due to this fact.

In table III are presented the results of the various methods of testing the infertile and fresh eggs. Out of 529 eggs examined, 64 were found to be positive by at least one of the methods used. Of these 64 that were positive; only five were positive by the direct smear procedure, a percentage of only 0.9. In the hatch of April 23, 2.6 per cent were positive, a figure that compares favorably with that of other workers using a comparable procedure. In the second procedure, namely transferring approximately 1 cc of yolk to brilliant-green, beef-extract broth and culturing on brilliant-green, liver-infusion agar after 48 hours of incubation of the egg yolk in the enrichment medium, the number of positive eggs was raised from five to six, giving 1.1 per cent positive. There appears to be little choice between these two procedures. Either one has little value and in no instance was a positive found by these methods that was not detected by procedures discussed later. The failure of these two procedures is attributable to the small amount of inoculum and the short periods of incubation used. In the third procedure,

TABLE III — *A comparison of various methods of culturing Salmonella pullorum from infertile and fresh eggs.*

Source of Eggs Examined	Eggs Showing Salmonella Pullorum								Positive	Negative	Totals
	Direct Smear		Brilliant Green Broth Enrichment		Dextrose Broth Enrichment						
					48 Hours		7 Days				
	No.	%	No.	%	No.	%	No.	%			
Hatched Apr. 12	2	2.2	2	2.2	4	4.3	7	7.5	8	85	93
Hatched Apr. 22	—	—	—	—	—	—	2	2.7	2	73	75
Hatched Apr. 23	2	2.6	1	1.3	8	10.3	9	11.5	9	69	78
Hatched Apr. 27	—	—	1	1.6	1	1.6	2	3.2	2	62	64
Hatched Apr. 30	1	1.2	2	2.4	10	12.0	14	16.9	14	69	83
Fresh	—	—	—	—	4	2.9	29	14.0	29	107	136
Totals	5	0.9	6	1.1	27	5.1	63	11.8	64	465	529

where the entire egg, both yolk and white, was introduced into 100 cc of dextrose, beef-extract broth, far better results were obtained. In this procedure, the broth was examined twice for the presence of *S. pullorum* by smearing brilliant-green, liver-infusion agar plates.

The first examination was made at the end of 48 hours, and the second after seven days of incubation. The results of 48 hours of incubation show 27 eggs positive (5.1 per cent), an increase from five eggs for the direct smear procedure to 27 for this method, and augmented efficiency of 540 per cent. This procedure shows the value of a large inoculum. When the enrichment broth was incubated for seven days, the number of positive eggs jumped from 27, for the 48-hour incubation, to 63, an increased efficiency over the direct smear procedure of 1,260 per cent. In only one instance did the 48-hour examination give a positive result when the seven-day observation failed. It would appear, judging from the data, that the latter method, with an incubation of the enrichment medium for seven days, would be the best procedure. The increase in the number of positives by incubating for seven days, over the results obtained after 48 hours of incubation, shows clearly the need of a lengthy incubation period to allow the organism time to overcome its long lag phase in growth. The results of a comparison of the procedures are presented in the graph in figure 1.

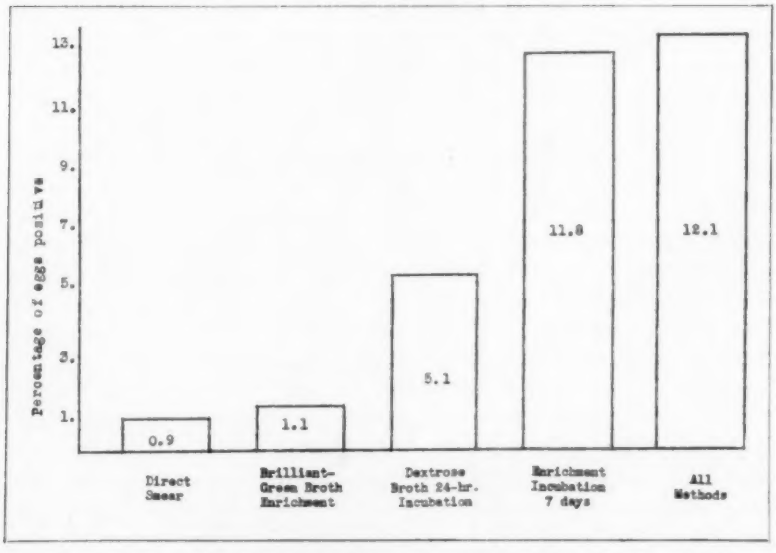

Fig. 1. Graph showing a comparison of various methods of culturing *Salmonella pullorum* from eggs.

The comparison of the three methods of examination of fresh eggs is interesting. The fresh eggs were incubated for 72 hours at 37° C. prior to testing for *S. pullorum*. The first two methods revealed no positive eggs, but when the eggs were incubated for seven days in enrichment medium, 29 eggs were found positive. The value of the long incubation period in the enrichment medium is also demonstrated quite strikingly. Incubation in the enrichment medium for 48 hours yielded only four positive eggs against 29 for the longer period of incubation. Of 136 fresh eggs examined by all methods, 21.6 per cent were positive. This percentage is particularly interesting, as it checks closely with the results obtained by Runnells and Van Roekel[6] on R. I. Reds and White Leghorns. Using a comparable technic, they found 20.1 per cent of the eggs from White Leghorns and 20.9 per cent of the eggs from R. I. Reds were positive. These three different flocks of reacting hens giving similar percentages would indicate that this percentage is probably nearer the incidence of the disease in fresh eggs than in the low percentages reported by Rettger,[2] Kaupp and Dearstyne,[9] Weaver and Weldin,[8] Gwatkin,[10] and Tittsler, Weywang and Charles.[7]

In table IV are presented the data for the incidence of *S. pullorum* in infertile eggs after incubation for ten days. Of 393 infertile eggs examined, 35 were found positive, a percentage of 8.9. The low incidence of disease in the infertile eggs, as contrasted to 21.6 per cent in the fresh eggs, is interesting. In the light of the discussion *pro* and *con*, that has appeared in the literature concerning the keeping quality of infertile eggs as compared to fertile eggs, these data have importance. It will be remembered that the infertiles have been incubated ten days as compared to three days for the fresh eggs. Hadley[11] showed that unincubated eggs showed 9.9 per cent bacterial infection as contrasted to 3 per cent for incubated eggs. It would appear that incubation either through the increased temperature or an increase in the natural antiseptic powers of the egg, caused by the higher temperature of incubation, may be responsible. This will be discussed more fully later in this paper. A wide divergence in the incidence of the infection in the different hatches occurred. The infection varied from a minimum of 2.6 per cent to a maximum of 16.9 per cent, with an average of 8.9 per cent of infertile eggs for the five hatches presented in the table. No explanation is offered for this variation.

TABLE IV—*The incidence of Salmonella pullorum in infertile eggs from reacting hens.*

DATE OF HATCH	EGGS EXAMINED	NEGATIVE	POSITIVE	
			No.	%
Apr. 12	93	85	8	8.6
Apr. 23	78	69	9	11.5
Apr. 27	64	62	2	3.1
Apr. 30	83	69	14	16.9
May 22	75	73	2	2.6
Totals	393	358	35	8.9

In table V are presented the data for the incidence of *S. pullorum* in the dead germs and chicks that died in the shell. Of the 241 dead germs and chicks dead in the shell, 124 were found to contain *S. pullorum,* a percentage of 51.4. In the individual hatches marked variations occurred. The hatch of February 19 showed 85.1 per cent of dead germs and chicks dead in the shell infected; whereas the hatch of April 30 showed only 24.3 per cent infected. As the season progressed, the number of positives decreased.

TABLE V—*The incidence of Salmonella pullorum in dead germs and chicks dead in the shell from reactor hens.*

DATE OF HATCH	EGGS EXAMINED	NEGATIVE	POSITIVE	
			No.	%
Feb. 9	34	10	24	70.5
Feb. 19	27	4	23	85.1
Mar. 3	21	6	15	71.4
Apr. 12	29	14	15	51.7
Apr. 23	40	22	18	45.0
Apr. 27	53	33	20	37.7
Apr. 30	37	28	9	24.3
Totals	241	117	124	51.4

In table VI are presented the data on the incidence of *S. pullorum* in the freshly hatched chicks. Of 236 chicks tested, 27 were found positive, a percentage of 11.4. In two hatches no infection was found, while in one hatch 20.4 per cent of the chicks were positive. As in the case of the dead germs and chicks dead in the shell, the incidence of the disease decreased as

the season progressed. This fall in the extent of the disease in the chicks at the time of hatching is in line with field experience with pullorum disease where it has been observed that the fatality of this disease in the chicks lessens as the season progresses.

TABLE VI—*The incidence of Salmonella pullorum in freshly hatched chicks from reactor hens.*

DATE OF HATCH	EGGS EXAMINED	NEGATIVE	POSITIVE	
			No.	%
Feb. 9	49	39	10	20.4
Feb. 19	61	53	8	13.1
Mar. 3	54	47	7	12.9
Apr. 12	20	19	1	5.0
Apr. 23	25	25	—	—
Apr. 27	10	9	1	10.0
Apr. 30	17	17	—	—
Totals	236	209	27	11.4

It is also interesting to note that in table VII, when the total number of eggs (infertiles, dead germs and dead in shell, and chicks) are considered in four hatches, the percentage of infection does not change. These data indicate that as far as the liveable chick is concerned, as the season progresses, the incidence of the disease becomes lower. Practically, late chicks should show considerably less infection; and further observations bear out this statement. Previous work by others is controversial.

Kaupp and Dearstyne,[9] reporting on fresh eggs, state:

> The per cent egg infection rises as production increases and reaches its greatest height at the season of maximum production.

The figures in table VIII are taken from their report. They are presented to show that the number of infected eggs rises as production increases, but the percentage of infection remains approximately the same. In fact, in March, the second highest month of production, the lowest percentages of infection were obtained in both flocks. The data presented show a constancy of infection rather than a rise during the period of high production as the authors claim. Runnells and Van Roekel[4, 6] present some very interesting data showing a fall in infection as the season advances. On an examination of fresh eggs, using a technic comparable to that used by the writers, they obtained the figures shown in table IX.

TABLE VII—*Summary of incidence of Salmonella pullorum in infertile eggs, dead germs and chicks dead in shell, and freshly hatched chicks from various hatches from reactor hens.*

Date of Hatch	Infertile Eggs			Dead Germs and Chicks Dead in Shell			Freshly Hatched Chicks			Totals		
	Negative	Positive		Negative	Positive		Negative	Positive		Negative	Positive	
		No.	%		No.	%		No.	%		No.	%
Apr. 12	85	8	8.6	14	15	51.7	19	1	5.0	118	24	16.8
Apr. 23	69	9	11.5	22	18	45.0	25	—	—	116	27	18.9
Apr. 27	62	2	3.1	33	20	37.7	9	1	10.0	104	23	18.1
Apr. 30	69	14	16.9	28	9	24.3	17	—	—	114	23	16.8
Totals	285	33	10.4	97	62	38.9	70	2	2.8	452	97	17.7

TABLE VIII—*Data on egg infection and production (Kaupp and Dearstyne).*

MONTH	REACTORS TESTED					
	24 RHODE ISLAND RED HENS			25 WHITE LEGHORN HENS		
	EGGS LAID	POSITIVE		EGGS LAID	POSITIVE	
		No.	%		No.	%
Nov.				6	1	16.6
Dec.	33	2	6.1	93	4	4.3
Jan.	121	9	7.4	143	7	4.9
Feb.	143	12	8.4	280	12	4.3
Mar.	221	6	2.7	392	11	2.8
Apr.	260	27	10.4	417	27	6.5
May	219	20	9.1	363	38	10.4
June	156	12	7.7	337	11	3.3
July	102	4	3.9	279	12	4.3
Aug.	58	4	6.9	195	8	4.1
Totals	1313	96	7.3	2505	131	5.3

TABLE IX—*Data on egg infection (Runnells and Van Roekel).*

DATE OF EXAMINATION	PERCENTAGE OF EGGS INFECTED	
	FRESH EGGS	INCUBATED EGGS
Feb.	32.6	32.4
Mar.	15.8	15.0
Apr.	7.4	7.8

These figures are particularly interesting because, in the case of the fresh eggs, they represent all of the eggs, without regard to fertility and in the second set of data for the incubated eggs they apparently represent only the infertiles, dead germs and chicks dead in the shell. These latter figures are somewhat comparable to those of the writers, given in table V, where the percentage of infection for the dead germs and chicks dead in the shell fell from 70.5 for the hatch of February 9, to 24.3 on April 30.

In table X are presented the data for the average incidence of *S. pullorum* in infertile eggs, dead germs and chicks dead in the shell, and freshly hatched chicks from reactor hens for the entire season. Although averages do not present the maximum and minimum conditions that have prevailed during the season, they

do show an interesting story of the prevalence of the disease in the egg under different conditions. It will be noted that the disease is unusually high in the dead germ and chicks that died in the shell, and low in the infertile eggs and the freshly hatched chicks. Inasmuch as the possibility of infection in the egg during the process of its formation is the same whether the egg is fertilized or not, would indicate that the percentage of infection has been higher in the infertile egg at some period previous to the time of testing. This would mean that some time after the egg had become infected in the hen, the organisms had been destroyed. As the fertile and infertile eggs were subjected to the same temperatures and methods of handling, the lower incidence of the disease in the infertiles would have to be attributed to some biological factor within the egg. That eggs do contain an antibiotic property has been demonstrated.

TABLE X—*The average incidence of Salmonella pullorum in infertile eggs, dead germs and chicks dead in the shell, and freshly hatched chicks from reactor hens for the entire season.*

SOURCE	EGGS TESTED	NEGATIVE	POSITIVE	
			No.	%
Infertile eggs	393	358	35	8.9
Dead germs and chicks dead in shell	241	117	124	51.5
Chicks	236	209	27	11.4
Totals	870	684	186	21.4

Rettger and Sperry,[2] working on the antibiotic action of egg white, demonstrated that when 500 *S. pullorum* were introduced into unheated egg white, no growth was obtained after exposure to the egg white for 92 hours. When the egg white was heated to 65-70° C. for 15 minutes, this antibiotic property was lost entirely. Other bacteria were also affected in a similar manner. The writers have been unable to find data demonstrating the presence of antibiotic action in egg yolk. However, the fact that *S. pullorum* occurs in the yolk and the fact that infertile eggs have such a low incidence of *S. pullorum*, the yolk must contain at least a selective inhibitory or germicidal activity for the organism. Furthermore, the fact that fertile eggs have a much higher incidence (33.7 per cent) of *S. pullorum* infection

than the infertiles (8.9 per cent) would indicate that fertile eggs, upon the development of the embryo or sooner, lose their antibiotic action. Practically, these data tend to confirm the general belief that infertile eggs keep better than fertile eggs, a view that has been argued ever since Gayon,[3] in 1873, presented his now historic work on the keeping quality of eggs.

It is rather difficult to arrive at a figure that represents the incidence of *S. pullorum* in the eggs of reacting hens.

The fact that the incubated eggs show an incidence of 21.3 per cent infection and the fresh eggs an incidence of 21.6 per cent does not indicate that the two methods of examination give comparable results and that the figure represents the actual incidence of infection. On the contrary, the marked variation between the incidence of 8.9 per cent (table IV) for the infertile eggs and 33.7 per cent (table XI) in the fertile eggs indicates that the percentage of 21.3 for those incubated is too low. To accept this figure it would be necessary to assume that at the time of laying only a few infertiles and a considerable number of the fertile eggs were infected. Such a theory is untenable. It can safely be hypothesized that all eggs, either fertile or infertile at the time of laying, contain the same incidence of *S. pullorum*. Thus the infertiles must have rid themselves to a considerable extent of the organism. These eggs can not be used in measuring the incidence of the infection in the egg. If we accept the fertile eggs as representing the incidence of the organism in this flock, the infection ranges from a maximum in one hatch of 40.9 per cent to a minimum of 16.6 per cent, giving an average infection of 33.7 per cent, a figure higher than any reported before.

It is possible, but difficult to prove, that at least some of the clean chicks may have developed from eggs that were infected at the time they were laid. If the total amount of infection in the fresh egg remains at an approximately constant level throughout the year, as evident from work previously cited, and the disease in the chicks from reactor stock drops off materially, as shown in this study and the work of Runnells and Van Roekel, it can be hypothesized that the embryo must throw off the disease in an increasing degree as the season progresses. This would lead one to believe that the actual incidence of the disease in the egg must be higher than the figure obtained for the fertile eggs and may approach the figure obtained for the infection in the dead germs and chicks dead in the shell. On this highly hypothetical assumption, the peak incidence would be the per-

TABLE XI—*The percentage of Salmonella pullorum infection in fertile eggs of seven hatches.*

Date of Hatch	Dead Germs and Chicks Dead in Shell			Chicks			Totals			
	Negative	Positive		Negative	Positive		Tested	Negative	Positive	
		No.	%		No.	%			No.	%
Feb. 9	10	24	70.6	39	10	20.4	83	49	34	40.9
Feb. 19	4	23	85.2	53	8	13.1	88	57	31	35.2
Mar. 3	6	15	71.4	47	7	12.9	75	53	22	29.3
Apr. 12	14	15	51.7	19	1	5.0	49	33	16	32.6
Apr. 23	22	18	45.0	25	—	—	65	47	18	27.7
Apr. 27	33	20	37.7	9	1	10.0	63	42	21	33.3
Apr. 30	28	9	24.3	17	—	—	54	45	9	16.6
Totals	117	124	51.4	209	27	11.4	477	326	151	31.7

centage infection obtained in the dead germs and chicks dead in the shell. In this study it would mean a maximum of 85.1 for one hatch and a minimum of 24.3 per cent, with an average of 51.4 per cent for the whole flock.

Even if we accept the more conservative estimates represented by the percentage infection of all the fertile eggs (33.7), the incidence of *S. pullorum* is higher than has ever been reported before. The work of Runnells and Van Roekel is the only one that approaches the data presented by the writers. As all previous workers, with the exception of Runnells and Van Roekel, failed to include the total number of infected eggs in their data, their results are not significant.

With the high incidence of *S. pullorum* in the eggs of reactors, it is interesting to note the small number of infected chicks that make up the final hatch. Out of four hatches from reactor hens in April (table VII) the percentage of infected chicks based on the total eggs set represented only 0.36. Thus the chance of a reactor hen introducing much infection through reactor chicks appears exceedingly remote. This is particularly true when the eggs of a commercial hatchery contain only a small percentage of reactors in the flocks supplying the eggs. The tremendous spread of the disease in the chicks is apparently not due to the large number of congenitally infected chicks, but to the highly contagious nature of the malady, and the extreme susceptibility of the clean chicks. The spread of the disease in the incubator has been stressed by Hinshaw, Moore and Upp,[14] Bunyea[15] and others. Its dissemination in the brooder by mixing chicks of reactors with clean stock, particularly under adverse temperatures, has been demonstrated by Moore, Mallmann and Arnold.[13] The importance of blood-tested stock, clean incubators, and favorable sanitary brooder conditions is again emphasized by this work.

SUMMARY

Infertile eggs from reacting hens show far less *Salmonella pullorum* infection than the fertile eggs from the same hens.

A study of eggs from reacting hens reveals that both dead germs and chicks dead in the shell show a much higher incidence of *S. pullorum* than the live chicks from the same hens.

The average incidence of *S. pullorum* in all the eggs from reactor stock is nearer that of the organism in the fertile eggs, rather than the average obtained by checking the presence of *S. pullorum* in both the fertile and infertile eggs.

The percentage of infection shown by chicks hatched from reactor stock is remarkably low. The extent of the disease in the chick is probably due, not to the large number of infected chicks hatched, but to the spread from a few infected chicks during hatching and brooding. Apparently pullorum disease is very contagious.

The need of segregation of clean stock, the immediate elimination of infected chicks, and the use of clean incubators and brooders is clearly demonstrated by this study.

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Conference on Fever Therapy

The First International Conference on Fever Therapy will be held at Columbia University, New York City, September 29-October 3, 1936. Abstracts of the papers constituting the program will be published in the proceedings in English, French and German. Further information may be obtained from Dr. William Bierman, General Secretary, 471 Park Avenue, New York, N. Y.

THE RESISTANCE OF SALMONELLA PULLORUM TO THE ACTION OF VARIOUS DISINFECTANTS*

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Few comparisons of the germicidal efficiency of the most widely used disinfectants against *Salmonella pullorum* have included tests with varying amounts of chicken manure, and practically no determinations have been made at temperatures other than 20° C. The phenol coefficient as determined by the standard Food and Drug Administration method may or may not give the true value of a disinfectant when used in the presence of organic matter and at a temperature other than that of the standard test. Since *S. pullorum* is disseminated through infected feces, and the disinfection of poultry-houses, brooders and incubators may be carried out in cold weather, determinations of germicidal efficiency were made with various amounts of chicken manure at 2, 20 and 40° C. These temperatures were used, as they approximate the maximum temperature range for tests with *S. pullorum:* below freezing there is a danger of pure crystals of water forming in the disinfectant solution and thus concentrating the disinfectant, while temperatures above 40° C. tend to reduce the resistance of this organism.

EXPERIMENTAL PROCEDURE

The F. D. A. technic for the testing of disinfectants[1] was followed, with the exception of the addition of suspensions of chicken manure and the use of three temperatures; 2, 20 and 40° C. The chicken manure suspensions were made as follows:

To a liter of water 400 grams of fresh chicken manure was added and mixed with a mechanical stirrer for 30 minutes, and then strained through a 16-mesh screen. The solid material was again mixed with a liter of water and, after similar stirring, was screened and the liquid added to the first liter. This constituted the "heavy suspension" while the "light suspension" was made by decanting a portion of the heavy suspension and screening through a 40-mesh screen. The suspensions of fecal material were autoclaved for 20 minutes at 15 pounds pressure. The pH of each suspension was 6.5. The heavy suspension yielded 2.8 gm of desiccated material and 0.25 gm of ash per 100 cc, while the light suspension yielded 1.235 gm of desiccated material and 0.27

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gm of ash. The material removed by screening from the two liters of material resembled wheat bran in appearance, weighed 32.42 gm and yielded 2.43 gm of ash.

A rapidly growing strain of *Salmonella pullorum* was selected which, after daily transfers for a week, gave plate counts of 204,000,000 to 260,000,000 with an average of 230,000,000 colonies per cc in 24-hour culture grown in F. D. A. broth. Where the effect of organic matter was not tested, 0.5 cc of culture was added to 5 cc of disinfectant; and where it was tested, equal parts of culture and chicken manure suspension were made, and 0.5 cc of the mixture was used as a culture. This reduced the number of organisms by one-half when the fecal suspensions were used, but did not materially alter the experimental results, since the rate of death of a bacterial population exposed to an unfavorable environment proceeds on a logarithmic rather than an arithmetical basis.

THE EFFECT OF CHICKEN MANURE SUSPENSION ON SALMONELLA PULLORUM

Since the fecal suspensions and the culture were mixed before adding the disinfectant, the effect of the suspensions on the organism is of interest. When held at 20° C., the results shown in table I were obtained.

TABLE I—*The viability of S. pullorum in chicken manure suspensions (culture 240,000,000).*

TIME	LIGHT SUSPENSION	HEAVY SUSPENSION
At once	95,000,000	101,000,000
1 hour	82,000,000	74,000,000
2 hours	66,000,000	70,000,000
4 hours	106,000,000	101,000,000
28 hours	133,000,000	104,000,000

Such variations could well be due entirely to random sampling, or they may indicate that during the first four hours a slight decrease in the number of viable organisms occurred, due to the changed environment; but the majority of organisms survived and the rate of increase slightly more than compensated for the death rate.

DISCUSSION

The data presented in tables II to VI show that the disinfectants may be divided in two classes. The phenol, the liquor

TABLE II—*The disinfection of Salmonella pullorum with phenol.**

TEMPERATURE	DILUTION OF PHENOL	ORGANIC MATTER ADDED								
		NONE			LIGHT SUSPENSION CHICKEN MANURE			HEAVY SUSPENSION CHICKEN MANURE		
		5	10	30	5	10	30	5	10	30
2° C.	1:40	—	—	—	—	—	—	—	—	—
	1:50	+	—	—	+	—	—	+	±	—
	1:60	+	+	—	+	+	—	+	+	—
	1:70	+	+	+	+	+	+	+	+	+
20° C.	1:70	—	—	—	—	—	—	—	—	—
	1:80	+	±	—	+	±	—	+	±	—
	1:90	+	+	—	+	+	—	+	+	—
	1:100	+	+	+	+	+	+	+	+	+
40° C.	1:150	—	—	—	—	—	—	—	—	—
	1:175	+	—	—	+	±	—	+	±	—
	1:200	+	+	—	+	+	—	+	+	—
	1:250	+	+	+	+	+	+	+	+	+

*Merck's reagent phenol used.
— = no growth. ± = growth in some tests, no growth in others. + = growth.

TABLE III—*The disinfection of Salmonella pullorum with liquor cresolis.**

TEMPERATURE	DILUTION OF LIQUOR CRESOLIS	ORGANIC MATTER ADDED								
		NONE			LIGHT SUSPENSION CHICKEN MANURE			HEAVY SUSPENSION CHICKEN MANURE		
		5	10	30	5	10	30	5	10	30
2° C.	1:60	—	—	—	+	—	—	—	—	—
	1:70	+	+	—	+	+	—	+	+	—
	1:75	+	+	—	+	+	—	+	+	—
	1:80	+	+	—	+	+	±	+	+	±
	1:100	+	+	+	+	+	+	+	+	+
20° C.	1:125	+	—	—	—	—	—	—	—	—
	1:150	+	+	—	+	—	—	+	—	—
	1:175	+	+	±	+	+	—	+	+	±
	1:200	+	+	+	+	+	±	+	+	+
40° C.	1:300	—	—	—	—	—	—	—	—	—
	1:350	—	—	—	+	—	—	+	±	—
	1:400	+	—	—	+	+	+	+	+	+
	1:500	+	+	+	+	+	+	+	+	+

*Liquor cresolis (U.S.P.), Smith, Kline and French, used.
— = no growth. ± = growth in some tests, no growth in others. + = growth.

TABLE IV—*The disinfection of Salmonella pullorum with formaldehyde.*

Temperature	Dilution of Formaldehyde	Dilution of Formalin	Organic Matter Added								
			None			Light Suspension Chicken Manure			Heavy Suspension Chicken Manure		
			5	10	30	5	10	30	5	10	30
2° C.	1:20	1:7.4	—	—	—	+	—	—	+	—	—
	1:25	1:9.2	+	—	—	+	+	—	+	—	—
	1:30	1:11.1	+	+	—	+	+	—	+	+	—
	1:40	1:14.8	+	+	±	+	+	+	+	+	+
20° C.	1:50	1:18.5	+	—	—	—	—	—	—	—	—
	1:60	1:22.2	+	+	—	+	—	—	+	—	—
	1:75	1:21.7	+	+	—	+	+	—	+	—	—
	1:100	1:37	+	+	—	+	+	—	+	+	—
	1:150	1:45.5	+	+	+	+	+	±	+	+	—
40° C.	1:150	1:45.5	—	—	—	—	—	—	+	±	—
	1:200	1:74	+	—	—	+	+	—	+	+	±
	1:250	1:92.5	+	+	—	+	+	±	+	+	+
	1:300	1:11.1	+	+	+	+	+	+	+	+	+

*Merck's reagent formaldehyde (neutral 37%) used.
— = no growth. ± = growth in some tests, no growth in others. + = growth.

TABLE V—*The disinfection of Salmonella pullorum with sodium hypochlorite.**

Temperature	Available Chlorine Parts Per Million	Organic Matter Added								
		None			Light Suspension Chicken Manure			Heavy Suspension Chicken Manure		
		5	10	30	5	10	30	5	10	30
2° C.	80	+	—	—	—	—	—	—	—	—
	60	+	±	—	+	—	—	+	±	—
	50	+	+	—	+	±	—	+	+	+
	40	+	+	+	+	+	+	+	+	+
20° C.	60	—	—	—	—	—	—	—	—	—
	50	±	—	—	±	—	—	+	—	—
	40	+	±	—	+	—	—	+	+	—
	30	+	+	+	+	+	+	+	+	+
40° C.	50	—	—	—	—	—	—	—	—	—
	40	+	±	—	+	—	—	+	—	—
	30	+	+	—	+	±	—	+	+	±
	20	+	+	+	+	+	+	+	+	+

*B-K used as source of sodium hypochlorite.
— = no growth. ± = growth in some tests, no growth in others. + = growth.

TABLE VI—*The disinfection of Salmonella pullorum with lye.**

TEMPERATURE	DILUTION OF LYE	ORGANIC MATTER ADDED								
		NONE			LIGHT SUSPENSION CHICKEN MANURE			HEAVY SUSPENSION CHICKEN MANURE		
		5	10	30	5	10	30	5	10	30
2° C.	1:750	—	—	—	—	—	—	+	—	—
	1:1000	—	—	—	+	+	—	+	+	—
	1:1250	+	±	—	+	+	+	+	+	+
	1:1500	+	+	+	+	+	+	+	+	+
20° C.	1:750	—	—	—	—	—	—	+	—	—
	1:1000	—	—	—	+	±	—	+	+	—
	1:1250	+	—	—	+	+	+	+	+	+
	1:1500	+	+	+	+	+	+	+	+	+
40° C.	1:1000	—	—	—	—	—	—	—	—	—
	1:1250	—	—	—	+	—	—	+	—	—
	1:1500	+	—	—	+	+	—	+	—	—
	1:2000	+	+	+	+	+	+	+	+	+

*Lewis lye, containing 98% NaOH used.
— = no growth. ± = growth in some tests, no growth in others. + = growth.

TABLE VII—*Recommended dilutions of various disinfectants at different temperatures.*

DISINFECTANT	2° C.	20° C.	40° C.
Phenol	1:10*	1:16	1:35
Liquor cresolis	1:13	1:25	1:80
Formaldehyde	1:5	1:10	1:40
Formalin (40% formaldehyde)	1:2	1:4	1:16
Chlorine (parts per million)	400	250	225
Sodium hypochlorite solution yielding 3.5% av. Cl.	1:87.5	1:140	1:155
Lye	1:225	1:250	1:300

*Greater than maximum solubility of phenol in water.

cresolis and the formaldehyde are markedly affected by the temperature, while the sodium hypochlorite and the lye are but slightly influenced. On the basis of the concentration required to kill in ten minutes, but not in five, which is the time interval suggested by the F. D. A. technic,[1] in the absence of added organic matter and at 2° C., phenol killed *S. pullorum* in a dilution of

1:50; liquor cresolis in 1:65; formaldehyde in 1:25; sodium hypochlorite, 70 parts per million of available chlorine, and lye in 1:1,175. Under these conditions the phenol coefficients are: liquor cresolis, 1.3; formaldehyde, 0.5; 285 for available chlorine or 10 for a 3.5 per cent solution of available chlorine, and 23.5 for lye.

The addition of chicken manure only slightly influenced the results; in the presence of light and heavy chicken manure suspensions at 2° C. the mean phenol coefficients are as follows: liquor cresolis, 1.35; formaldehyde, 0.45; available chlorine, 333, and lye, 15.75. It is interesting to note that the sodium hypochlorite solution was as effective in the presence of chicken manure as in its absence. This phenomenon has been reported previously.[2]

At 20° C., in the absence of added organic matter, phenol killed *S. pullorum* in 1:80; liquor cresolis in 1:125; formaldehyde in 1:50; sodium hypochlorite in a concentration of 50 parts per million of available chlorine, and lye in 1:1,250. In the presence of the heavy suspension of chicken manure, lye killed in 1:750. At this temperature the mean phenol coefficients are: liquor cresolis, 1.77; formaldehyde, 0.71; available chlorine, 250, and lye, 12.5.

At 40° C. a 1:175 phenol dilution and a 1:400 liquor cresolis dilution killed *S. pullorum* in the absence of chicken manure, while a 1:350 dilution killed when it was present; formaldehyde (1:200) killed in the absence of chicken manure and a 1:150 dilution killed when it was present; sodium hypochlorite, 40 parts per million, killed, and lye killed in dilutions of 1:1,250 to 1:1,500. The mean phenol coefficients at 40° C. are: liquor cresolis, 2.1; formaldehyde, 1; available chlorine, 143, and lye, 7.6.

CONCLUSIONS

Official phenol coefficients obtained against *Eberthella typhi* at 20° C. may give little information as to the comparative values of these agents used in the disinfection of poultry-houses and brooders against *Salmonella pullorum*. Since concentrations five times that necessary to kill in ten minutes, but not in five, are recommended to provide a satisfactory margin of safety, the dilutions to be used at the various temperatures are given in table VII.

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A NOTE ON THE INFLUENCE OF CULTURE MEDIA ON THE ISOLATION OF PASTEURELLA FROM EQUINE CEREBRAL TISSUE OF ARTIFICIALLY INOCULATED HORSES*

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During the fall and winter of 1934-35, an acute epizoötic encephalomyelitis-like disease of horses, commonly referred to as "cornstalk disease," occurred in central Illinois. Carefully computed estimates indicate that approximately 5,000 horses died of this disease, entailing a loss of $500,000. The clinical symptoms observed in affected horses resembled the virus type of encephalomyelitis,[1-4] as reported during recent years in eastern and western states. Notwithstanding the clinical analogy, more than 50 equine brain specimens were injected into guinea pigs, pigeons, white mice, and horses, in an effort to demonstrate the presence of a filtrable virus or virus-like agent. Negative results were obtained.[5]

Epizoölogically, so-called cornstalk disease of horses differs from the filtrable virus type of encephalomyelitis of horses in that the former occurs during winter. Gross and microscopic pathology of the encephalon in so-called cornstalk disease also affords differential characteristics. In more than 50 brains examined, 65 per cent showed gross cerebral lesions of softening, similar, if not analagous, to the areas of cerebral liquefaction noted by MacCallum and Buckley[6] more than 30 years ago. Histopathologic changes described in the encephalon of the filtrable virus type of equine encephalomyelitis disease were not encountered in cornstalk disease. On two previous occasions, so-called cornstalk disease has been reported in epizoötic proportions in Illinois. Each of these outbreaks (1893 and 1914), as well as the 1934-35 outbreak, followed a summer drouth and heavy fall rains which resulted in an abundance of worm-eaten, moldy inferior corn.

NATURAL CASES OF CORNSTALK DISEASE

In a bacteriologic study of the brains of horses that died of so-called cornstalk disease (toxic encephalitis or non-virus encephalomyelitis or encephalomalacia), cerebral tissues were seeded on solid and liquid culture media. The purpose of this report is to call attention to the results on these two types of

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media, with special reference to the propagation of Pasteurella-like types, as well as the results of culturing the brains of horses that died following artificial Pasteurella infection.

The cerebral tissues selected from natural cases for inoculating both types of media were often obtained at the junction of the white and gray matter incident to a deep longitudinal incision of the cerebral hemisphere, which was made primarily for the purpose of noting gross pathologic lesions. In cerebrums showing gross areas of liquefaction, the tissue selected for inoculating culture media originated adjacent to the gross lesions and were seeded directly on plain agar, 10 per cent horse-blood agar, and gentian-violet agar plates. Following incubation, the inoculated plates often showed a profuse growth of a variety of bacteria and mold colonies. Diplococci and streptococci predominated, while staphylococci, *Escherichia coli* and other unidentified bacteria were frequently encountered. Discreet, sparsely scattered, small colonies of Gram-negative rods, often staining at the poles, frequently developed on the inoculated plates. These colonies on transfer repeatedly failed to grow. Pasteurella[7] isolations in liquid media inoculated with brain-tissue specimens invariably yielded negative results on solid media when directly seeded with the brain tissue. The non-viable colonies noted on solid media might therefore have been Pasteurella types, though this conjecture has been neither proved nor disproved. In eight specimens,[8] Pasteurella-like microörganisms, as judged by cultural characteristics on differential media, were isolated. One Pasteurella was obtained by streaking the liquid cultures of the medulla on solid media, although inoculation of laboratory animals (pigeons, guinea pigs and rabbits) with the impure liquid cultures of the medulla and cerebrum has often yielded Pasteurella strains. In another fatal case of equine cornstalk disease (clinical illness 10 to 12 hours duration, accompanied by liquefaction of the cerebrum), Pasteurella was regained from the heart-blood by culturing in liquid media and transplanting to solid media.

Experiment Horses

Negative and positive results with reference to Pasteurella on the two types of culture media seeded with the same brain tissue specimens of naturally affected horses prompted the intravenous inoculation of three healthy horses with saline suspensions of *Pasteurella equi*-like microörganisms for the purpose of culturing the brains following death, on liquid and solid media. The object of the protocol was to compare the results obtained in the liquid

and solid culture media heretofore mentioned in the isolation of Pasteurella from the encephalon of known infected horses.

The horses selected for these experiments had not been fed corn for many months and at no time had they received corn that was raised in 1934. In so far as the feeding of damaged 1934 corn was related to so-called cornstalk disease, the test horses used in these studies were free from any dietetic corn factor. The inoculum was grown on 10 per cent horse-blood agar, suspended in sterile physiological sodium chloride solution and adjusted to McFarland tube 5. One horse received a Pasteurella-like microörganism (20380) regained from the heart-blood of a guinea pig that had been inoculated with a saline suspension of a mixed culture from the corn-meal mash cultures of a horse medulla (table I). The two remaining horses received a Pasteurella-like microörganism (12175A) isolated from a guinea pig that died following feeding of worm-eaten, moldy corn. Both strains displayed cultural, morphological and tinctorial characters as well as carbohydrate fermentation reactions ascribed to the Pasteurella group.

TABLE I—*Intravenous inoculation of Pasteurella.*

HORSE	CULTURE	DOSE	RESULTS
23070	Artificial Pasteurella Infection 12175A GP 839 HB	2 20-cc	
		1 30-cc	Died in 6 days
23078	Artificial Pasteurella Infection 12175A GP 839 HB	1 20-cc	Died in 24 hours
26384	Artificial Pasteurella Infection 20380 GP 410 HB	2 20-cc	Died in 5 days

BACTERIOLOGIC RESULTS IN INOCULATED HORSES

Immediately following death, each horse brain, enclosed in the meninges, was removed from the cranium with aseptic precautions. After the removal of the meninges, a deep longitudinal incision was made into the cerebrum, and small pieces of cerebral tissue, from the junction of the gray and white matter, were streaked on the surface of plain and gentian-violet agar plates by means of sterile forceps. Simultaneously small pieces of cerebral tissue were placed in tubes of meat mash, corn-meal mash, and brain mash. All inoculated culture media were incubated for 72 to 96 hours or longer at 37° C. and examined for evidence of growth. The liquid brain, meat, and corn-meal mash cultures, following incubation at 37° C. for 48 to 72 hours, were transferred with sterile platinum loop to plain and gentian-violet agar plates.

The plain and gentian-violet agar plates inoculated from the cerebral tissues of horses 26384, 23078, or 23070 remained sterile. However, Pasteurella-like strains were regained from meat-mash and brain-mash, cerebral-tissue-inoculated cultures of horse cerebrums 26384 and 23078 when they were transplanted to plain and gentian-violet agar plates. Similar results were encountered in the corn-meal-mash, cerebral-tissue-inoculated cultures of horse cerebrum 23078 and brain-mash and corn-meal-mash cultures of equine-cerebral-tissue-23070-inoculated cultures by streaking on gentian-violet agar plates (fig. 1). The three strains isolated were identified by morphological and tinctorial characters as well as by carbohydrate fermentation reactions. Acid was produced in dextrose, saccharose, galactose, inulin, levulose, mannitol, sorbitol and xylose, while no acid was noted in lactose, maltose, arabinose, raffinose, rhamnose, trehalose, salicin and dextrin.

Discussion

Since a subclinical Pasteurella infection in laboratory animals may be excited to fatal termination by a variety of causes, the advantage of more accurate direct cultural methods in the diagnosis of cerebral as well as other types of pasteurellosis is apparent. On the other hand, it is recognized that isolation of Pasteurella by direct cultural methods from animal tissues cannot be accepted as conclusive diagnostic evidence of a primary pasteurellosis until all other factors are excluded.

The cultural results herein reported indicate that liquid media, such as 10 per cent corn-meal mash, brain mash, or meat mash, are more suitable than solid media for the isolation of Pasteurella and Pasteurella-like microörganisms. These observations also suggest a possible explanation of the negative results secured on solid media in routine diagnostic procedures heretofore employed in the cultural diagnosis of Pasteurella *via* animal inoculation in seeding tissue from the parenchymatous organs of laboratory animals following death, which have been inoculated with tissue suspensions from animals showing acute hemorrhagic septicemia-like symptoms.

In this connection, a possible parallel in pathogenic bacteriology may be mentioned in the field of human pathology in the isolation of meningococci from the cerebrospinal fluid of man, wherein a cultural diagnosis of meningococcus infection is more accurately accomplished by inoculating flasks of dextrose broth or other suitable liquid, serum-enriched media with centrifugalized spinal fluid, and after a period of incubation transferring the resultant growth to solid media for identification.

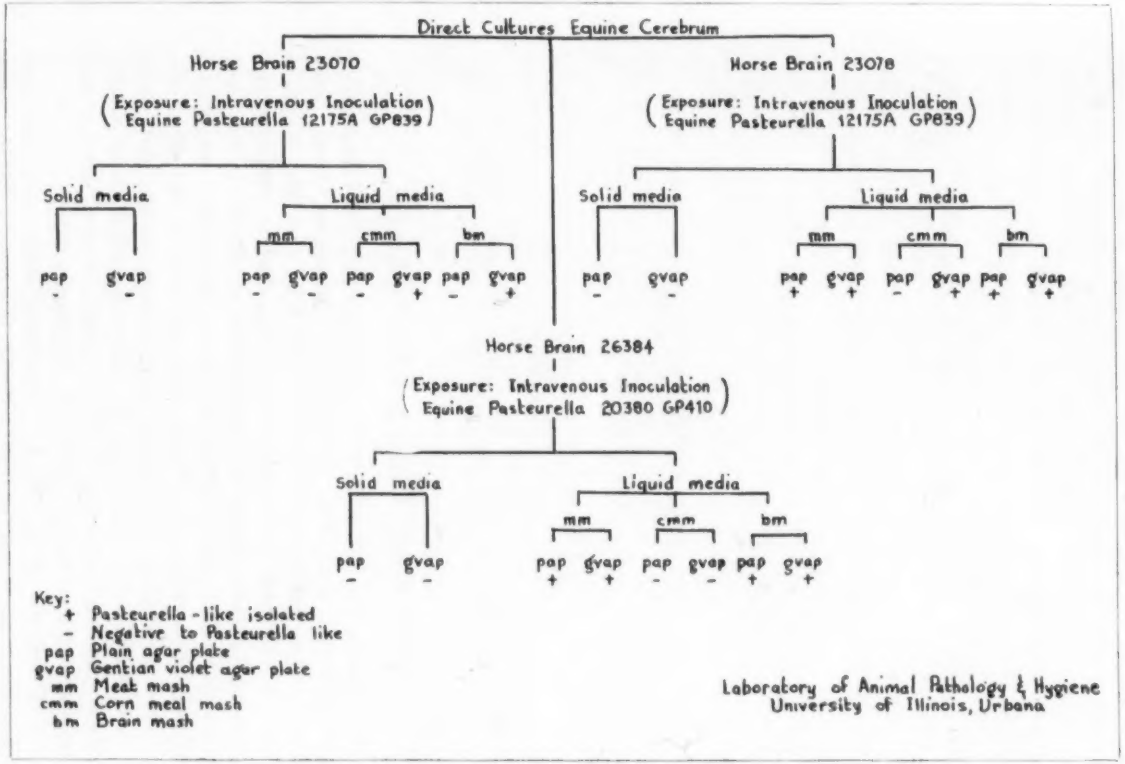

FIG. 1. Graph showing results of inoculations in studies on so-called cornstalk disease (equine)—toxic encephalitis.

SUMMARY

The results of cultural studies on equine cerebral tissues obtained from horses immediately following death from artificial Pasteurella inoculation suggest that the type of media selected for direct propagation of Pasteurella strains is important in accurately appraising the presence or absence of a cerebral Pasteurella infection of the equine encephalon.

Different types of liquid media discussed, upon being inoculated with bits of cerebral tissue, yielded positive results in all three horses, while the same cerebral-tissue specimens heavily seeded on plain agar and gentian-violet agar plates proved negative.

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Bruce Boyce Takes London by Storm

Bruce Boyce, son of Dr. Walter Boyce, a U. S. Bureau of Animal Industry inspector, stationed at Superior, Neb., gave his first London recital at Grotrian Hall, May 27. Mr. Boyce, a baritone of decided accomplishment, presented a wide-ranging program containing songs by Schubert and Wolf, as well as modern composers. According to a London critic, the control of a remarkably attractive voice never faltered, and in the diversity of moods represented, Mr. Boyce left no doubt of his skill, both as a craftsman and an interpreter. The songs had been clearly visualized and were presented with a shapeliness of phrase and climax that testified to an alert and responsive mind.

Dr. T. P. White, A. V. M. A. resident secretary for England, attended the concert and writes that Mr. Boyce took his audience by storm and received as many curtain calls and encores as a seasoned artist. It is predicted that the young baritone is headed for grand opera and will travel fast and far.

PARATYPHOID INFECTION IN TURKEYS*

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The epizoötics of paratyphoid in turkeys which constitute the subject matter of this paper occurred in 1934 on ten Iowa and one southern Minnesota turkey farms.

HISTORICAL

Turkey production in the state of Iowa has been greatly increasing during the last twelve years. Prior to 1922, turkey raising in Iowa was considered more or less of a side issue to regular live stock farming and only a few turkeys were raised on a few farms each year, principally for local use. At the present time, there are more than 500,000 turkeys raised annually on Iowa turkey farms, with many people turning to turkey farming as a main enterprise. While a few eggs for hatching are obtained locally, most of them come from California, Texas, Nebraska and South Dakota.

PARATYPHOID EPIZOÖTIC OF 1934

Prior to 1934, turkey farmers in Iowa had unusual success in raising turkeys as far as losses from disease were concerned, these running from 1 to 3 per cent annually. These losses were due principally to enterohepatitis, parasitism, injuries and faulty diet.

In May, 1934, our attention was first called to a highly acute disease present in young poults under five weeks of age. The mortality was extremely high, reaching as much as 90 per cent in some flocks in the five-week period. The first farm visited (1) had approximately 10,000 birds of two different hatches, of about twelve and three weeks of age, respectively. Losses had not occurred in the birds twelve weeks of age but were occurring at the rate of about 1,000 per week in the birds three weeks old, and during the first five-week period amounted to 70 per cent. The onset of the disease was very sudden and apparently was not checked by careful handling and sanitation. Farms II, III and IV presented a very similar picture and were of the same hatch as the birds three weeks old on farm I. Farm V had rather poor equipment and the sanitation and management were rather poor. There were about 6,000 two-week-old birds on this farm. They were not

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from the same hatch as those on the farms previously mentioned and the loss on this farm over a six-week period was about 40 per cent.

Farms VI, VII and VIII were not visited, but birds from them were sent to the laboratory for examination and the same condition was found present, but the losses on these farms were not so great. The source of these poults was not the same as that of the previously mentioned flocks. The eggs were obtained from local breeding flocks which were not in good condition and a great deal of roup existed among the birds. As soon as trouble started in the young poults, the breeding flock was disposed of and eggs from Texas were used. Very little trouble was experienced in subsequent hatches.

Farms IX and X are located in the northwestern part of the state and were not visited, but birds were received from them at the laboratory for diagnosis. The poults on these farms were from eggs shipped from Nebraska. The loss on farm IX was about 60 per cent and on farm X about 90 per cent of 3,000 poults.

Birds from one farm in Minnesota were shipped to the laboratory for diagnosis and were found to be suffering from the same condition as encountered on Iowa farms. No history as to percentage of losses could be obtained. The total loss in Iowa attributed to this disease in 1934 was estimated to be about 10 per cent of 350,000 birds. The disease made its appearance again in a similar manner in 1935 but losses were not so great, the total loss on the ten farms of the 1934 outbreak being 2 per cent of 385,000 birds. There were several outbreaks on farms not affected in 1934, with losses as high as 60 per cent.

SYMPTOMS

The symptoms were very much the same on all farms where the disease occurred. The affected poults frequently appeared to be chilled, and crowded close to the hover where they remained. Diarrhea was noted at times but was not a constant symptom. Death was sudden in many cases. Poults appearing normal in the morning died during the day or, appearing to be normal in the evening, were found dead in the morning. In those cases where death was not so sudden, the poults were weak, unthrifty, with ruffled feathers, dragging wings, slow and sluggish movements, and a thin, watery gaseous diarrhea. The birds would sit or stand for long periods with eyes closed, head drawn back under the wing or hanging forward on the breast, remaining very quiet without the usual cheep or call of healthy poults.

Postmortem Appearance

While the macroscopic pathologic picture was not constant, the most pronounced lesions of infected poults were: inflammation of the duodenum, varying from mild to severe; congestion of the liver, kidneys, gall-bladder and heart muscle. In a great many cases the pericardial sac was filled and distended with a serous, straw-colored fluid. The bone-marrow in a majority of cases was hyperplastic. The ceca varied from a mild to a great distention, filled with a coagulated cheesy-like material composed principally of feed, fecal material and epithelial débris, very much resembling cecal plugs sometimes found in young chicks suffering from *Salmonella pullorum* infection. There was but a slight amount of inflammation present and no hemorrhage into the cecal contents. The most constant lesions found both in birds posted at the laboratory and in birds killed and posted on the farms visited were inflammation of the duodenum, congestion of the liver, kidneys and heart muscle, and presence of a coagulated cheesy mass of material in the ceca. The lesions observed were very similar to those previously described by Rettger, Plastridge and Cameron.[5]

Histopathologic Findings

Microscopic study of the various tissues by means of tissue sections revealed the following changes in tissues or organs: hyperemia of the liver, varying from slight to rather intense, with occasional hemorrhage into the liver tissues; mild hyperemia of kidneys and heart muscle. Sections of bone-marrow were not entirely satisfactory but showed some evidence of a slight hyperplasia. Section of the ceca did not show as extreme a condition as upon gross examination. The microscopic lesion was marked by a partial or complete desquamation of the cecal epithelium, with various zones of inflammatory reaction.

Bacteriologic Findings

A paratyphoid-like organism was isolated from birds in the first group of three-week-old poults from farm I. Isolation was made from heart-blood, liver, spleen and bone-marrow on liver-infusion agar and heart-infusion agar. Upon first examination of the cultures from both tubes and plates it was believed that there was more than one variety of organism. However, systematic study failed to establish any definite characteristic differences between them, and they were regarded as belonging to one definite species. The organism is a motile, Gram-negative, non-

sporulating rod which does not liquefy gelatin. It varies considerably in length but is definitely somewhat shorter than *Salmonella pullorum*. It stains quite readily with ordinary aniline dyes. It grows readily in ordinary culture media such as meat-infusion agar, plain agar, peptone broth, meat broth and liver agar. However, growth is more luxuriant on liver-infusion agar, with a distinct tendency toward pleomorphism, a feature not observed on other media. It ferments dextrose, galactose, levulose, maltose, mannitol, rhamnose, dulcite and mannose, with production of acid and gas. It does not attack saccharose, dextrin, inosite, raffinose, salicin, inulin and lactose, and xylose is attacked very slowly, with only a small amount of gas formation. It browns lead acetate, does not produce indol, reduces nitrates to nitrites, and does not produce acetyl-methol-carbinol. The colonies on liver-infusion agar plates appear smooth and glistening, gray to white in color. Broth becomes turbid with a slight amount of gray sediment forming. Litmus milk becomes slightly acid and later alkaline. The same species of organism as above described was isolated from birds on all the farms studied.

Serologic Properties

Agglutination tests of the blood of birds in the breeding flocks which supplied eggs for farms I, II, III and IV were impossible, since most of the eggs were from flocks in California and Texas. The local breeding flocks which had supplied some eggs early in the season had been destroyed because of the extremely low hatchability of the eggs.

The breeding flock on farm V, which numbered 251 birds, was available for testing. The method used was the standard tube method, using dilutions of 1:25, 1:50 and 1:100. The antigens used were made from various organisms of the Salmonella group, namely, *S. gallinarum*, *S. pullorum*, *S. anatum* (secured from Dr. Rettger), *S. aertrycke*, turkey strain A1 (secured from Dr. Rettger), and turkey strain isolated from farm I in Iowa. The antigen was made by growing the above organisms for five days on liver-infusion agar, washing off and diluting with phenolized saline solution to a density between tubes 1 and 2 of the McFarland nephlometer. Cross-agglutination between the different strains occurred in high dilutions, thus rendering the simple agglutination test inadequate as a basis of differentiation of the organism in question. The test, however, would have some value as a test for breeding flocks, for the purpose of elimination of diseased birds. The results of the tests with the various strains

TABLE I—*Agglutination reactions, farm V.*

BIRD	S. PULLORUM	S. GALLINARUM	S. ANATUM	TURKEY A1 —RETTGER	TURKEY 1 —IOWA
2	100	100	0	100	100
4	50	50	0	100	100
5	50	50	0	50	50
6	100	50	0	50	50
7	100	100	0	100	100
11	100	100	0	100	100
13	100	100	0	100	100
14	100	100	50	100	100
16	100	100	0	100	100
21	100	100	0	100	100
22	100	100	0	100	100
30	100	100	50	100	100
36	50	50	25	100	100
44	50	50	0	50	50
45	50	50	0	50	50
46	50	50	0	50	50
57	100	100	0	100	100
59	50	50	50	100	100
60	100	100	0	50	50
64	50	50	50	0	25
68	50	50	50	100	100
71	50	50	50	0	0
72	50	50	50	50	50
75	50	50	0	50	50
76	100	100	0	100	100
53	0	0	0	100	100
78	100	100	0	100	100
80	100	100	0	50	50
81	0	0	0	100	100
85	0	0	0	100	100
87	100	100	0	0	0
91	50	50	0	25	25
92	100	100	0	100	100
96	100	100	0	100	100
97	0	0	0	100	50
99	100	100	0	0	0
106	100	100	0	0	0
111	50	50	0	100	100
115	0	0	50	100	100
116	0	0	0	100	100
118	0	0	0	50	50
119	100	100	0	0	0
138	100	100	0	100	100
139	100	100	0	100	100
145	50	50	50	50	50
146	25	25	25	25	25
153	0	0	25	100	100
154	100	100	0	100	100
170	100	100	0	100	100
172	50	0	50	50	50
196	100	100	0	100	100
204	0	0	0	100	100
231	100	100	0	100	100
243	50	50	0	50	50
185	0	0	0	100	100
190	50	50	0	50	50

on those birds showing reaction is given in table I. The remainder, not shown in the table, were negative to all strains used.

Only nine birds from a previous breeding flock of 100 on farm VI were left. Samples secured from these nine survivors were tested, using antigens prepared in the same way as for the test in table I, the only difference being in the organisms used.

Agglutination experiments were conducted with serum of rabbits immunized against the following organisms: *Salmonella schottmülleri* (Jordan), *S. aertrycke* (Rettger), *S. anatum* (Rettger), *S. pullorum*, *S. gallinarum* and organisms isolated from turkeys on farms I, II, III, IV, V and VI. The following technic was used for immunizing the rabbits. Forty-eight-hour cultures on liver agar were used and intraperitoneal injections were made at five-day intervals. With a sterilized 4-mm platinum loop, 2 loopfuls of culture were mixed into 4 cc of sterile saline solution. The emulsion was heated for 30 minutes at 60° C. in a water-bath, then cooled and injected. The second dose was similarly prepared except that 4 loopfuls were used in 4 cc of saline solution. The third dose contained 6 loopfuls in 4 cc of solution,

TABLE II—*Agglutination reactions, farm VI.*

BIRD	TURKEY STRAIN I	TURKEY STRAIN II	TURKEY STRAIN III	SALMONELLA PULLORUM	SALMONELLA ANATUM
1	0	0	0	0	0
3	0	0	0	0	0
4	50	50	50	0	0
5	0	0	0	0	0
6	0	0	0	0	0
9	100	100	100	100	0
13	100	100	100	0	0
14	100	100	100	100	100
16	0	0	0	0	0

the fourth dose one agar slant in 5 cc of saline solution, and the fifth and sixth doses one agar slant in 5 cc of saline solution. The fifth and sixth doses were not heated and were live cultures. The blood was tested ten days after the last injection and the titre noted. There was a great variation in titre, running from 1:6,400, in the case of strains of turkey organisms, to 1:400 with *S. schottmülleri*. Great difficulty was encountered in this method of immunization due to deaths of rabbits from injection of live cultures or to toxins produced. In several cases animals died after the last injection and before the agglutinin titre was at any height. The method of detoxification with formaldehyde, em-

ployed by Batchelder[1] with *Pasteurella pestis*, is far more satisfactory and was used with very good results. Cross-agglutination between the different strains, with the exception of *S. anatum*, was evident. The agglutinin-absorption method of Castellani, as employed by Edwards and Rettger,[2] was next employed with the different strains and absorbed serums were quite definite, so we were able to identify the turkey paratyphoid strain as *Salmonella aertrycke*.

Pathogenicity for Chicks, Turkey Poults, Guinea Pigs, Rabbits and Adult Turkeys

Chicks: A group of 36 chicks, eleven days old, was used for the first experiment. The chicks were hatched in the laboratory from eggs of a pullorum-free breeding flock. Fifteen of these chicks received intraperitoneal injections of 0.5 cc of a heavy saline suspension, corresponding to McFarland tube 5, of a 24-hour culture grown on liver-infusion agar. Another group of 15 chicks was given orally, by means of a pipette, 1 cc of a 24-hour culture suspended in saline solution. The six other chicks were used as controls. The first group of chicks died during the first 36 hours and the organism was recovered from the heart, liver and spleen in all cases. The second, or orally infected group, died between the fifth and twelfth days, most losses occurring between the fifth and eighth days. The organism was recovered from the heart, liver and spleen in all but one case.

Lesions in artificially infected chicks: There were no definite lesions in the first group other than hyperemia of the liver and a slight evidence of a septicemia in a few chicks. In the second group those birds dying during the first period of losses, that is, from the fifth to the eighth day, showed a severe hyperemia of the liver and kidneys and a severe duodenitis. Those dying from the eighth to the twelfth day showed hyperemia of the liver, kidneys and heart muscle, hyperplasia of the bone-marrow, and distention of the ceca, with a cheesy, coagulated mass, very similar to that observed in field cases. A severe duodenitis also was present. The six chicks used as controls remained healthy during the twelve-day period. This experiment was repeated later, using 21-day-old chicks with quite similar results. In adult chickens mortalities occurred in 60 per cent of the injected chickens in a period of ten days, but those fed suspensions of the organisms failed to develop symptoms and no fatalities occurred.

Turkey poults: Thirty-six turkey poults, five days old, were divided into three groups. Group 1, consisting of 15 poults, received intraperitoneally 0.5 cc of a 24-hour liver-infusion agar

culture suspended in saline solution, corresponding to McFarland tube 5 in density. Group 2, consisting of 15 poults, received 1 cc of the above suspension orally by means of a pipette. The six poults in group 3 were used as controls. The poults in group 1 started dying during the first 24 hours and were all dead at the end of four days. In all cases the paratyphoid organism was recovered from the heart-blood, liver and spleen. The poults in group 2 receiving the organisms orally died as follows: three on the fifth day, five on the sixth and seventh days, three on the twelfth day and one on the 25th day, four birds surviving the infection. In all cases that died paratyphoid organisms were recovered from the heart, liver, spleen, or intestine, except from two birds, one of which was so badly decomposed that no examination was made. There were no lesions except hyperemia of the liver in the first group. Those birds in group 2 that lived over seven days showed lesions very similar to those found in field cases on farm I, particularly duodenitis and the presence of coagulated plugs in the ceca. The six control poults remained healthy throughout the experiment.

Guinea pigs: Six guinea pigs weighing about 200 grams each were used. These were divided into three groups of two each. Group 1 was injected intraperitoneally with 1 cc of a saline suspension of a 24-hour culture of the paratyphoid organism in saline solution. Group 2 was given 2 cc of the same suspension orally, group 3 being used as controls. The two guinea pigs in group 1 died during the first 24 hours, with a severe septicemia. The paratyphoid organism was recovered from the heart-blood, liver and spleen. One guinea pig in group 2 died on the sixth and the other on the ninth day. The paratyphoid organism was recovered from the heart and liver of both animals. An appreciable loss of weight was observed in both these animals. The lesions in group 2 were hyperemia of the liver with a few small pin-point areas of necrosis, duodenitis and hyperemia of the kidneys. The spleen appeared somewhat swollen and gray in color, with a definite increase in connective tissue evidenced by cutting through the organ. Group 3, used as controls, remained healthy for 21 days, and then were killed, but no lesions were observed.

Rabbits: An experiment was conducted on eight adult rabbits. They were divided into four groups of two each. Group 1 was injected intravenously with 0.2 cc of saline suspension corresponding to McFarland tube 5 of the paratyphoid organism grown on a liver agar slant. Group 2 was given 0.5 cc of the same suspension intraperitoneally. Group 3 was given 3 cc of the same suspension orally by means of a pipette. Group 4 served as con-

trols. The two rabbits in group 1 died in the first 36 hours, with a septicemia, and the paratyphoid organism was recovered from the heart-blood, liver and spleen of both subjects. Of the two rabbits in group 2, one died on the tenth day and the other on the 21st day and the organism was recovered from both animals by culturing the heart-blood, liver and spleen on liver-infusion agar. The two that were given cultures orally remained alive and showed no ill effects but did show an agglutination titre of 1:50. This experiment was repeated, giving 5 cc instead of 3 cc with similar results. The controls remained in good health. Lesions observed in rabbits were those of a septicemia in the first group, plus hyperemia of the liver and kidneys with a slight duodenitis in group 2. A few small necrotic areas were present in the liver of one animal in group 2 but it was not definitely determined whether this was due to the paratyphoid infection.

Adult turkeys: Five adult female turkeys and one tom over one year of age were secured for inoculation purposes. The agglutination test was first applied, using an antigen made with *S. pullorum* and one with the turkey paratyphoid organism, but with negative results. Four hens were given orally 5 cc of a saline suspension corresponding in density to McFarland tube 5 of the paratyphoid organism from 24-hour cultures made on liver-infusion agar. This was continued for ten days and the only noticeable symptom was a profuse diarrhea occurring in one hen on the tenth day. It was very noticeable that in spite of a good ration the birds lost weight during 15 days as follows: bird 1, from 13 pounds to 10 pounds; bird 2, from 12 pounds to 10 pounds; bird 3, that developed a diarrhea, 13 pounds to 9 pounds; bird 4, from 9 pounds to 8 pounds. At 28 days, agglutination tests were applied, using antigen prepared from cultures of the turkey paratyphoid organism. Bird 1 showed reaction in 1:50; bird 2, 1:100; bird 3, 1:25; bird 4, 1:100. The birds were destroyed 30 days after inoculation and a complete autopsy and cultural examination was made. Lesions consisting of a mild duodenitis, hyperemia of the liver and kidneys and severe inflammation of the ovaries were very evident in turkeys 1, 2 and 4. The ovaries of bird 1 showed a yellow-green discoloration with hyperemia. Turkey 3 was suffering from enterohepatitis, which possibly accounts for the severe diarrhea shown by the bird. The paratyphoid organism was isolated from the ovary of turkeys 1 and 4 and from the liver and intestine of turkeys 1, 2 and 4. Turkey 3 was not cultured. Isolation from the intestine was

made on Endo medium with the aid of the preliminary enrichment medium of Browning, Gilmour and Mackie.[3]

Transmission Experiments

Because the organism was recovered from the ovary of infected turkey hens and because the age incidence of the disease in young poults was somewhat similar to that of *S. pullorum* infection in chicks, the following experiments were undertaken.

Ten turkey hens and two toms were purchased from farms V and VI. These birds had previously shown an agglutination reaction of 1:100 or above. Each of these birds was fed 5 cc of a suspension of the turkey paratyphoid organism suspended in saline solution of a density of McFarland tube 5 for ten days. Agglutination tests were made after 30 days and all birds showed agglutination of 1:100 or above. The birds were then housed and eggs collected daily. A total of 60 eggs was secured from this flock and set in an incubator at the laboratory. From this setting forty poults were hatched. These were placed in a brooder-house, using a coal stove for heat. Two poults died the first night and cultures of the heart-blood, liver and bone-marrow were negative. The remainder of the birds did very nicely and showed a reasonable growth until the tenth day, when a number of them appeared chilled and remained under the hover constantly. They showed ruffled feathers, dragging wings and loss of appetite. Two of the poults died during the night, six more the next day, five on the 12th day, four on the 13th day, and seven on the 14th day. The losses continued until about the fourth week, at which time 30 poults had died. The lesions in the birds that died were very similar to those described from field cases on farms visited, which were hyperemia of liver and kidneys, duodenitis and a filling of the ceca with a coagulated mass of material. The organism isolated appeared to be the same as that from field cases and was pathogenic for guinea pigs, rabbits and young chicks. It was recovered from the liver, heart and bone-marrow of twelve of the 30 poults that died. The ten hens from which the eggs came were destroyed and carefully autopsied and cultured. The paratyphoid organism was isolated from the ovaries of four of the ten. This organism was also pathogenic for rabbits, guinea pigs and young chicks.

Blood Studies

Blood studies during the course of paratyphoid infection in turkeys were not highly successful because of the irregularity and rapidity of deaths in experimentally infected poults and the

lack of symptoms in older birds. However, in those studied it was observed that an increase in eosinophiles took place very early after inoculation and the total white cells showed an extreme increase above normal. Monocytes were observed early in the onset of the disease, later decreasing. While phagocytic action on the part of leukocytes in bacteriemia of birds is as yet not proven, it would seem probable that the tendency shown by the trend of white cells in paratyphoid infection might be indicative of exerted function.

Cultures from Eggs

Turkey eggs were cultivated in an effort to isolate the causative organism. The method of Bushnell and Mauer[4] was employed. The causative organism was not isolated from the eggs cultured. Only 81 eggs were available for this study, and of this number, only eight could definitely be traced to reactor hens. Many of the eggs cultured had been incubated from ten to 29 days. Possibly positive results could have been obtained if a larger number of eggs had been available. This statement is based on the fact that *S. aertrycke* was later isolated from the ovaries of reactor hens.

Alcaligenes faecalis was isolated 10 times, *Aerobactor aerogenes* 11 times and *Pseudomonas convexa* 3 times. Members of the Staphylococcus genus were isolated 14 times, *Bacillus subtilis* 12 times and *Bacillus cereus* 6 times. Twenty-eight eggs were sterile. The eight eggs coming from reactor hens were in the latter group. In considering the organisms isolated one must consider the age of the eggs at the time they were cultured.

The incubator humidifier was studied as a possible source for spreading the disease. Samples of water were taken from the humidifiers at various times during the hatching season. *Escherichia coli* was isolated in every instance. *Salmonella archibaldi* and *Aerobactor oxytocum* were each isolated once. Day-old poults were fed and inoculated with suspensions of *S. archibaldi*. This organism proved to be non-pathogenic for poults.

Summary and Conclusions

1. A paratyphoid organism was isolated from turkey poults of various ages under five weeks from widely distributed areas in Iowa.

2. Large losses were experienced by the owners, the greatest losses occurring in poults under five weeks of age.

3. The incidence of occurrence on the same farms was very low in the second year.

4. A general systematic study of the organism including the application of the simple agglutination test as well as the agglutinin-absorption test identified the organism as *Salmonella aertrycke* type.

5. The organism is highly virulent for baby chicks, young poults, guinea pigs and rabbits when administered orally, intraperitoneally or intravenously.

6. Newly hatched poults may spread the infection to others. The cycle of infection seems to be very similar to *Salmonella pullorum* infection in chickens.

7. The symptoms are weakness, unthriftiness, loss of appetite, ruffling of feathers, dragging of wings, sleepiness, diarrhea, sudden onset and high mortality.

8. The lesions of the disease in turkey poults may be said to be duodenitis, hyperemia of the liver and kidneys, hyperplasia of bone-marrow of the long bones, a distention and filling of the ceca with a cheesy, coagulated plug of fecal material, undigested feed material and epithelial débris.

9. The blood picture presents a leukocytosis with a large degree of monocytosis.

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Iowa Membership in the A. V. M. A.

Year	*Members*	*Year*	*Members*
1923	269	1930	298
1924	249	1931	291
1925	308	1932	290
1926	291	1933	261
1927	277	1934	231
1928	260	1935	208
1929	280	1936	?

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

SOME OBSERVATIONS ON THE EUROPEAN BROAD FISH TAPEWORM DIPHYLLOBOTHRIUM LATUM*

By J. E. THOMPSON, *Ely, Minnesota*

The fish tapeworm, *Diphyllobothrium latum*, has received little attention in veterinary literature. What mention is made of it tends to picture it as a freak and this is very likely due to its rare appearance over the country at large. Man and animals contract the infestation through eating raw or poorly cooked, fresh-water fish. Previous to 1927, it was contended that such cases of man or beast found harboring this parasite contracted the infection by eating fresh-water fish of European origin and it was assumed that the life-cycle was not carried on in American lakes or streams. During that year, Ward, Essex and Magath,[1] working at Ely, Minnesota, proved that the life-cycle was carried on in American waters, and it was my good fortune, through the local Board of Health, to be of some assistance to these men. This city and vicinity was found to be an endemic area, and having practiced here for over 15 years, I have gained some knowledge of this parasite in the dog and humans as well. Since 1927, I have examined feces for the local hospital, and have come to the conclusion that the literature and the local frequency of the parasite in animals and humans must be studied together.

In our veterinary literature, one of the early reports of this parasite is by Hall and Wigdor,[2] who observed it in a dog at Detroit. Van Es and Schalk[3] also reported finding the parasite in a dog at Fargo, and believed that they had traced the infection to fish of oriental origin. In April, 1927, Dr. Hiram E. Essex[4] came to Ely to make preparations for the investigation which took place during the following summer, and in my collection of parasites, he observed a specimen of the broad fish tapeworm. I was able to give him the history of the case, as it was from one of my own dogs, to which I had been feeding lightly cooked fish which came from the lake into which our city's sewage is discharged. Previous to this, Dr. Essex had evidence of a fish tapeworm found by our local physicians in a two-year-old child,[5] and this, with the history of my dog case, appeared to make certain that the life-cycle of the parasite was carried on in our vicinity. In 1928, Vergeer[6] made some investigations in Minnesota, and his reports minimize the human as the spreader of this parasite, and fixed the factor to a greater extent on the dog.

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Description and Life-Cycle

The ova of the fish tapeworm are egg-shaped and yellow in color, with a cap-like structure on one end. (Fig. 1.) This is the only tapeworm egg of domestic animals which does not have six hooklets at the time it passes from the host. The inner structure of the egg will show fine, shot-like bodies. The eggs will hatch in water within a few weeks, the length of time required depending on the temperature. The embryonic worm escapes from the egg through the end-cap, becoming a larva which is propelled by cilia. Certain forms of small crustacean life known as cyclops will then engorge the larvae and they become the first intermediate host. Six species of Cyclops were found in Shagawa Lake, but only two of these, namely, *C. strenuus* and *C. prasinus* were effective as the first intermediate host. These cyclops are next swallowed by a small fish. The cyclops is digested by the fish, and the small larva of the fish tapeworm is released. It has undergone further development, having several saber-like appendages with which it cuts its way through the intestines

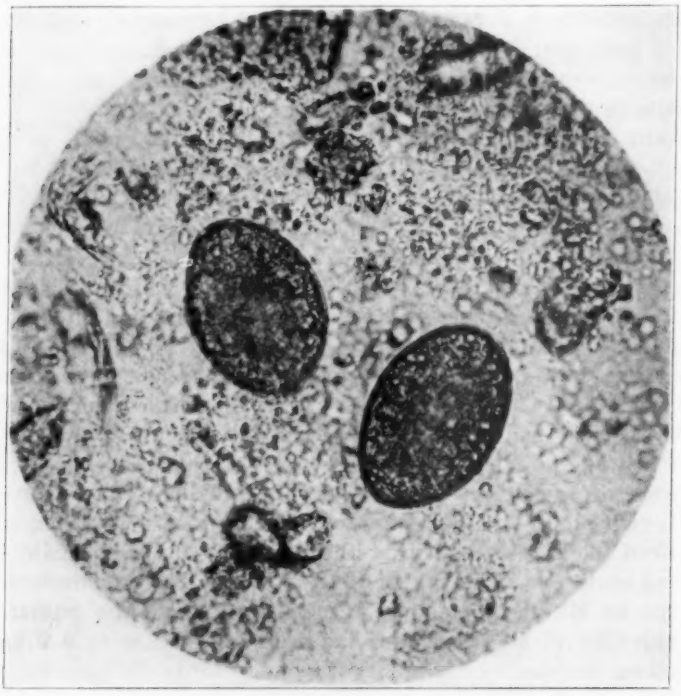

Fig. 1. Ova of *Diphyllobothrium latum* in feces of dog (x 410).

and penetrates into the flesh, and thus the fish becomes the second intermediate host. The larval worm coils up and lodges itself to await some mammal to consume it and permit development to the adult stage of the tapeworm. In searching fish for larvae or plerocercoids, one must skin the fish and steak it into thin bacon-like strips.

The adult worm, when found in man, obtains a length of 25 feet, and the segments have a maximum width of ⅝ inch. The head is very small and has two slit-like suckers with which the parasite attaches itself to the intestine. The feature which will most easily differentiate *D. latum* from other tapeworms is the brownish spot on each segment, giving a beaded appearance throughout the length of the parasite. (See figure 2.) These spots are the ovaries. The width of the segments is greater than their length. The tail segments break off, rupturing the ovaries and discharging the ova into the intestinal contents. When found in a dog, the tapeworm is dwarfed in length and breadth as compared with cases where man is the host. Likewise, Essex found the ova of canine origin to be less than 10 per cent hatchable, while the ova in human feces are nearly 100 per cent hatchable. From these observations, it appears as if man is the preferred or true host of the adult tapeworm.

In 1927, a four- to five-pound northern pike, taken from Shagawa Lake at Ely, could be expected to show 20 to 40 larval worms or plerocercoids, and the pike, northern pike and perch were 100 per cent infested in greater or lesser extent. (Figs. 3 and 4.) Such fish as crappies, suckers and mongrel whitefish, which also inhabit this lake, have at no time been found to harbor this larval stage of the parasite. Annually, since 1927, I have had occasion to examine the species of fish known to harbor the larval worms, and have observed a decreasing amount of infestation per fish, but still, each fish can be expected to have one or more plerocercoids. The same species of fish, taken from lakes upstream and downstream in this chain of lakes, shows the larva in small numbers. It is evident that Shagawa Lake is receiving the ova in great numbers with the sewage discharged from the city of Ely.

The frequency with which the tapeworm, *D. latum*, is found in humans in this vicinity is noteworthy. The local physicians will treat ten or more cases a year. I have known of many other people harboring the parasite to treat themselves with remedies purchased of local and advertising druggists. Almost 100 per cent of the human cases are confined to one nationality, the

Finns, and the Finnish newspapers are the only newspapers wherein I have observed druggists advertising a tapeworm remedy for humans. In the course of examining human feces, I have found one case of tapeworm other than the broad fish tapeworm. The few cases where it has been found in other nationalities can usually be accounted for in that such people have had access to the brine-pickled fish, a Finnish national dish.

I have found the broad fish tapeworm to be the most common tapeworm in the dogs of this vicinity. The fish are seldom fed to the dog. Instead, they gain access to it at garbage heaps and places where fishermen clean their catch. Most dogs will refuse raw fish, but once they have acquired the taste, their owners frequently report the dog as refusing his regular diet,

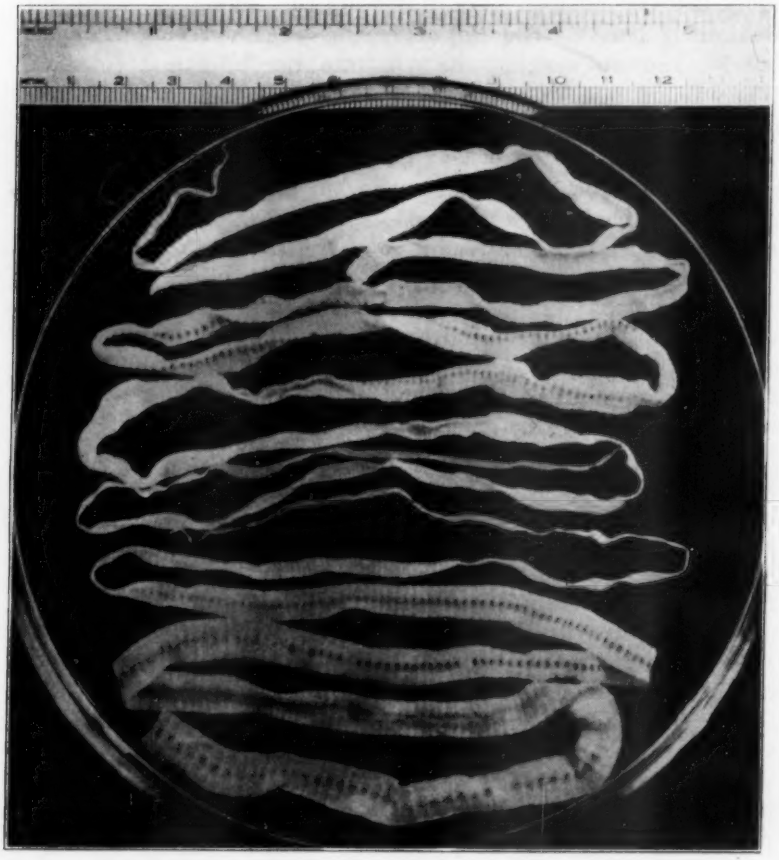

FIG. 2. *Diphyllobothrium latum* (adults) from dog, Cedar Rapids, Iowa, 1934.

and shortly thereafter he will be found eating fish at a garbage pile. Such dogs will often be reinfested after treatment. One of the striking observations in connection with this parasite in both the human and dog cases is that as a rule the host will harbor only one of these tapeworms. However, an experiment dog, kept in my kennels, was fed eight pieces of raw fish, a larval worm in each piece, and after four weeks, when the feces of the dog showed the ova, it was killed, and in the small intestine were found eight broad fish tapeworms about 20 inches long. I have frequently fed cats pieces of raw fish with the parasite, but in all my years of practice, I have not observed one cat with this tapeworm. Thinking that the feeding habits of the cat

FIG. 3. Plerocercoids of *Diphyllobothrium latum* in pike muscle.

might destroy the plerocercoid, I fed my own cat three pieces of fish with the larval worm by poking them down his throat, and even this gave negative results. The bear and other wild animals have been reported as being hosts to this tapeworm.[7]

EFFECT ON HOST

In most cases, whether in man or dog, this parasite causes little clinical disturbance. However, in young dogs in particular I have observed decided evidence of malnutrition accompanied by a ravenous appetite. In one case of a three-year-old child, I

did observe malnutrition and an anemic appearance. A few exceptional cases in man have been observed at Ely with very marked anemia. One such case showed a red cell count of 2,000,000, and this individual was subject to fainting spells. Cameron[8] states the anemia to be due to a hemolysin liberated by the parasite. He states also that tapeworms are heavy calcium consumers and take their nourishment by osmosis from the food within the intestinal tract. *D. latum* is the only tapeworm with which anemia is associated. All symptoms disappear rapidly upon removal of the parasite.

FIG. 4. *Diphyllobothrium latum* plerocercoid from pike muscle (x 12).

In older dogs the parasite seldom produces any serious symptoms. The owner usually reports having seen the tapeworm hanging from the anus, and he immediately brings the animal for treatment. Many humans have knowingly carried this fish tapeworm for years and have not bothered about taking treatment. Sometimes they pass considerable portions of it and break it off with their fingers and let it go at that. At one time I was present when a man sitting in a privy called for his wife to bring a pair of scissors; the rest can be pictured by yourself.

TREATMENT

In considering the effectiveness of a vermifuge for this tapeworm, one must not be satisfied with merely removing a quantity of the worm. It frequently happens that the head or scolex is retained and regeneration takes place. Likewise, it is difficult to remove the parasite intact, the head very often breaking off and, being so small, it is difficult to find in the feces. Whenever possible, I like to make a recheck of the feces about six weeks after treatment.

Our local physicians like to hospitalize these patients so as to make certain of proper administration of the remedy and to

observe the results. They prefer to have the patient fast before treatment and precede the vermifuge with a saline purgative or cascara. Fluid extract of aspidium is given in large doses, and when preceded by the foregoing treatment there is less vomiting due to the nauseating effect of the male fern.

In dog cases, I prefer to have them miss a meal or two before treatment, and then administer arecoline hydrobromide. Until a few years ago, I used ¼- to ½-grain doses, but of late years I have felt that 1/10-grain doses give better results. It seems to me that in my dog cases I get too many regenerations due to a retained scolex, whereas, my physician friends seldom have the same trouble.

One factor which should not be overlooked in making examinations of the feces for the ova of this tapeworm is that it is possible to have two or more successive stools without any ova, and then find a positive one. The tail segments may not always be developed to the stage that they discharge ova. Likewise, in human cases, more than in dog cases, there can be considerable periods during which no clinical evidence of the parasite is observed, either as segments or an actual portion of the tapeworm protruding from the anus. During such periods, what apparently happens is that the tail segments break off and are disintegrated and lost in the feces. For two years, I had occasion to take fecal samples from one man for experimental purposes. He did not know how long he had the worm, and seldom had he observed any clinical symptoms. His feces continually showed the ova in great numbers. Without any treatment the ova disappeared from his feces and they continued to remain negative.

Control Measures

In considering control measures for a parasite, we aim to strike at some point in the life-cycle where, with mass treatment, we can prevent further progress of the life-cycle. In the case of *D. latum*, as sanitarians or sportsmen, we should desire such control to be accomplished. It would appear offhand that the fish in Shagawa Lake at Ely are the primary source of this infection, but this is not the case. Lake trout are the fish which most commonly are eaten as a brine-pickled fish, and trout do not inhabit this lake. Lakes which do have this fish are located at some distance from the city. Among the human cases of this infestation are found the principal fishermen of the community, and they will make excursions of considerable length to obtain their choice fish. It has been claimed that dogs spread the infestation to these remote lakes, but dogs very rarely get to such

places. The fishermen usually travel with canoe, and one can readily picture how they leave their feces along the immediate shore lines to have the rains wash the ova into the lakes and streams. Shagawa Lake has gotten into ill repute because of Ely's sewage, and is seldom fished by anyone.

The present sewage treatment plant at Ely was put into operation in 1926. It is an Imhoff tank, the plans of which were approved by the Division of Sanitation of the State Board of Health, so it can be considered as being of proper size and construction, and an example of the average type of sewage system. Previous to the year 1926, we had a septic tank entirely too small for the volume of sewage to be dealt with, and raw sewage did enter the lake. Since 1928, I have tried to determine if the ova of *Diphyllobothrium latum* will pass through this Imhoff tank. Hall[9] states that sewage disposal systems and privies provide safe disposal of human feces and thereby become barriers to the continuance of the life-cycle of many parasites.

The volume of sewage to be treated by the Imhoff tank at Ely varies between 300,000 and 500,000 gallons daily. From my observations as to the operation of sewage disposal plants, I can state that the Imhoff tank at Ely receives at least the average attention. The influent sewage readily shows the presence of *Diphyllobothrium* ova in dipped samples, but they are not found so simply in the effluent. In order to make a more thorough study of the effluent, the following steps were taken:

First, the engineering department determined the period of the day during which the peak volume of sewage must be handled and this was found to be between 9 and 10 a. m. Effluent sampling was done three hours later by dipping small quantities at short intervals, so that within one hour a gallon of sewage would be collected. A sample of the influent sewage was taken between 9 and 10 a. m. The reason for this difference in time of taking samples is that the Imhoff tank has a minimum retention or displacement period of three hours, and this sampling method gives us a cross-section of the sewage going in and coming out.

The sewage samples, after being well shaken, were poured into two cone-shaped graduates of one liter capacity, calibrated at the bottom to tenths of a cubic centimeter. The samples were then allowed to stand for 24 hours. During this time, the solid material in suspension settled to the bottom of the graduates. The difference in the amount of solids in suspension in the influent and effluent samples is an indication of the efficiency of

the sewage treatment plant. Over a period of days, the influent samples varied between 4 and 12 cc of solids, and the effluent samples between 0.2 and 0.8 cc. With a long pipette, the settled solids were drawn from the graduate containing the effluent sample, and this material was searched for ova under the microscope. Out of 20 days of sampling, one ovum was found in each of two daily samples. This indicates that the Imhoff tank will not eliminate the ova but will materially reduce their numbers. Theoretically, if there were no solids in suspension in the effluent sewage, no ova should be found, but such efficiency of this type of sewage disposal plant is difficult to obtain. When we consider the volume of sewage in comparison with the amount tested, it is readily seen that there are still thousands of ova going through the tank. We can here raise the question: If human feces were deposited about the landscape much as those of animals are, would as many ova as the reduced numbers guided speedily by our sewerage system escape the hazards of topography and the elements and safely reach a lake or stream in hatchable condition? Having shown that ova can go through a sewerage system, we can cite additional evidence in the young fish of the lake which are 100 per cent infected. One can hardly picture the ova which infect these fish as having entered the lake prior to 1926, when the Imhoff tank was put in operation.

There are various factors which cut down the efficiency of a sewage treatment plant; one of them can be cited here. Most cities have two sewerage systems, namely, sanitary sewers and storm sewers. The storm sewers should receive street drainage and roof drainage, but we frequently find roofs of many buildings drained into sanitary sewers. This type of connection, during a heavy rainfall, causes a large increase in volume of liquids to be handled by the sewage treatment plant, thereby cutting down the retention period. During such times, no doubt, an increased number of parasite ova will pass through. Sanitary engineers have felt for some time that more thorough sewage treatment is needed. They base their opinions as to the efficiency of a sewage plant on the physical, chemical and bacteriological nature of the effluent. Let us hope parasite control will likewise be considered.

Conclusions

1. Man is the chief, and the dog a lesser, factor in the spreading of the broad fish tapeworm.

2. Since ova of *Diphyllobothrium latum* can go through an Imhoff tank, the presence or absence of parasite ova in a sew-

erage system effluent should be used as an additional index in judging the efficiency of such plants.

3. Thorough cooking of fish is the best safeguard against the parasite.

ACKNOWLEDGMENT

The author wishes to express appreciation to Dr. E. A. Benbrook, of Ames, Iowa, for his help and photography.

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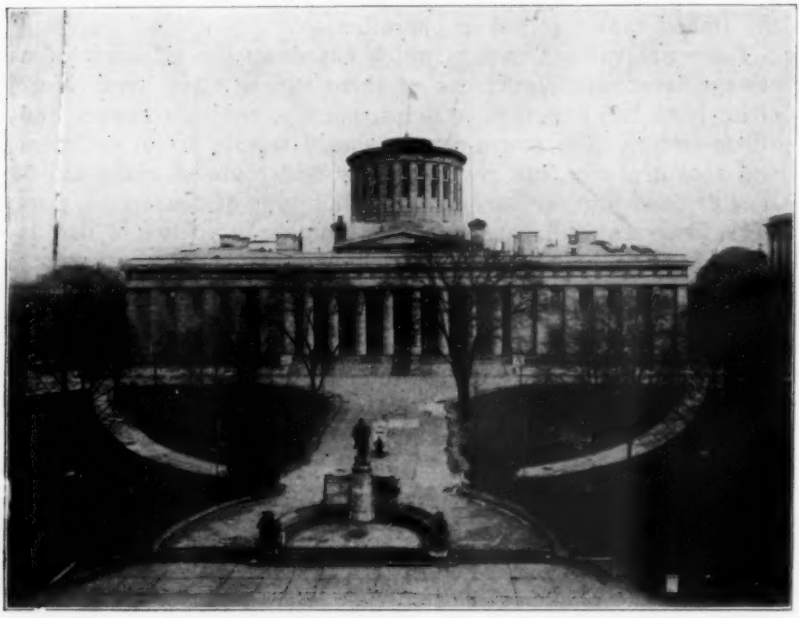

STATE CAPITOL, COLUMBUS

SALT DEFICIENCY IN HERBIVORA*

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On March 3, 1936, I was called to attend a mule and arrived at the farm about 6:15 p. m. Upon my arrival, the animal was standing perfectly still, in a "dummy" attitude with the feet spread somewhat in saw-buck fashion. The animal had been wet with perspiration, which is characteristic of animals just prior to death. Life's activities were so feeble that they were hardly perceptible. The eyes were staring and would not react to stimuli; the jaws were set and the tongue was paralyzed. The mouth appeared to be full of dirt. I did not take the temperature of the animal as I did not expect it to live, of which I notified the owner almost immediately after seeing the patient.

With reference to the weather conditions, it was a beautiful sunshiny day, but not very hot. The barometer was rather high, as was the humidity. I made some rapid deductions: The animal had already gone through the so-called death sweat, was suffering from sunstroke and was greatly dehydrated. The animal had been standing up all winter and suddenly had been taken out for work in the sun all day. The body was low in salt content, otherwise the so-called death sweat could not have occurred. When I started treatment, there was almost a cessation of the vital functions due to a further deranged mineral balance brought about principally by a shortage of salt in the body over quite a period of time.

TREATMENT

The first medication given the mule was a subcutaneous injection of 3 cc of a 4 per cent solution of potassium permanganate in 17 cc of a saturated solution of equal parts of common salt and baking soda. The injection was made high up on the neck above the jugular groove. An intramuscular injection of 10 cc of sterile camphorated oil was given, as well as a subcutaneous injection of 90 cc of dextro calci-nate (Jen-Sal). At the end of ten minutes, the heart was pounding. Next, the animal was

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given a rectal injection of about four gallons of water containing two small handfuls of salt. By this time, I was able to put about one tablespoonful of salt in the animal's mouth.

Recovery

Gradually the animal was able to move its tongue slightly. Then the eyes took on a more rational appearance. The nose began to twitch and it was drawn to the left, showing that the left side of the brain was more affected than the right. The heart action gradually subsided to normal. The animal was able to move its head up and down a little, then the nose became normal except for a greatly dilated nostril, and later the nostril became normal.

When I found that the animal could open its mouth, I rinsed it out with some water and also put some salt in it. Then the animal began to move its feet slightly. Previous to this, the animal had been insensible to stimuli. At 7:30 p. m., the animal was able to go to the trough and walked into its stall in a nearly normal fashion. I would not allow it to drink all the water it wanted, but limited the amount to two buckets. I placed another small handful of salt in the feed-trough and noted that the animal was exceedingly salt hungry. Therefore, I had a large tub of water put in the stall that night. The mule was given a feed of oat hay, as it was hungry. I took my leave at 8:00 p. m., confident that the animal would recover. I returned early the following morning and found the animal acting normally, but still very salt hungry. I advised feeding one tablespoonful of salt daily until the extreme salt hunger had subsided and advised keeping rock salt before the animal at all times when it was in the barn.

Conclusions

This is a case of retarded mineral metabolism brought about primarily by lack of salt. This opinion was substantiated by the owner who stated that he had given his mules no salt for two weeks. Herbivora eat entirely of the vegetable kingdom and are plentifully supplied with potassium, magnesium, calcium, phosphorus and the volatile compounds. When salt is withheld for any length of time, sodium and chlorine are missing. These two elements fill the gap in the electro-chemical exchange of elements in the presence of colloids, and without the two elements metabolism stops.

In order to start the metabolism of the elements again, the two fixed alkalies, potassium and sodium, must be supplied immediately and in the right proportions. Chlorine must also be supplied in order that it may combine with potassium to be elim-

inated by way of the urinary tract. Sodium must be supplied in abundance for perspiration, and from a practical standpoint, the cessation of kidney action means a lack of potassium, and the loss of water from the body suddenly by way of the skin denotes an extreme shortage of sodium compounds.

ARTHRITIS IN A CHICK CAUSED BY SALMONELLA PULLORUM*

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On June 6, we received two dead Single Comb White Leghorn chicks, about three weeks of age, from Passaic County, N. J. No history accompanied the specimens. The one specimen showed a large blood-clot in the pericardial sac and another in the thoracic cavity. The other specimen was of interest by virtue of a somewhat fluctuating swelling at the tibio-metatarsal joint on each leg.

The swelling was opened and a lemon-colored, gelatinous material expressed. There were no other visible changes in the body. The gelatinous material yielded a pure culture, which produced acid and gas in dextrose and failed to ferment lactose, maltose or saccharose. An antigen prepared from the culture was completely agglutinated at a 1:160 dilution of serum from a carrier bird, and partially in a 1:320 dilution. Simultaneously, a known *Salmonella pullorum* antigen was agglutinated completely at 1:40 and partially at 1:80. Serum from a control bird failed to agglutinate either antigen in a 1:20 dilution. It would appear then that this was a case of arthritis provoked by *S. pullorum* infection.

The case is of further interest because a similar condition is of common occurrence in pigeons. In this species the wing joints are usually involved and the condition is referred to by laymen as "lumps." Investigators are usually in accord in reporting *S. aertrycke* as the causal agent. However, Emmel[1] claims to have isolated *S. schottmulleri*, a closely related organism. A good description of this type of disease has been given by Brunett,[2] who found the swellings "filled with a straw-colored, viscid material."

The finding of *S. pullorum* in a joint constitutes a new point

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of localization to be added to the growing list. A review of the points of localization may not be out of place here.

Although the ovary is most commonly the seat of localization, other organs should not be disregarded. Jones[3] was the first to call attention to nodules in the myocardium, with gelatinous exudate in the sac. Rettger *et al*[4] encountered cases of pericarditis in males in which yolk-like masses were found in the sac with a clear amber-colored fluid. From two of eleven reacting males examined by them, the organism was recovered from the testes. Knight[5] also recovered the organism from the testes of a reacting cock bird. From nine reacting males, Doyle[6] recovered the organism in four instances from the testes, twice from the spleen, once from the testes and gall-bladder, once from the spleen and testes and once from a necrotic area in the heart muscle. In a previous study, Doyle[7] had recovered the organism from the ovary in 37 cases out of 42 reacting fowls. In the same series, the organism was found in the spleen in eleven cases, in the gall-bladder wall in nine, in the kidney in four, in the liver of two, and in the bile once. Rettger *et al*[8] isolated *S. pullorum* from a thoracic abscess in a reacting hen whose ovary was normal. Hitchner[9] recovered the organism from nodules in the mesentery. In a case reported by Bushnell *et al*,[10] the ovary yielded a culture, as did tumor-like abscesses on the skin of the posterior part of the abdomen and legs. The organism was isolated by Rettger[11] from an abdominal cyst in a male.

Brunett[12] has called attention to the frequent occurrence of nodules in the wall of the muscular stomach, from which the organism can be isolated. The diagnostic value of these as well as nodules in the lungs is now generally recognized. Finally, Beaudette[13] isolated the organism from the middle ear of a chick showing torticollis.

In summarizing, this brief review of the literature indicates that *S. pullorum* may be isolated, as a localized infection, from the ovary, testes, spleen, liver, gall-bladder, thoracic abscess, myocardium, pericardial sac, skin, middle ear and, finally, the joint cavity.

Addendum

After the above was prepared, the note by Durant and McDougle[14] was discovered, in which they describe identical cases in two different flocks. These authors point out that infections might cause lameness in chicks as well as such deficiency diseases as slipped tendons, rickets, etc. This raises the question as to

the possible part played by a deficiency disease in favoring localization of an infection in the joint.

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GRANULOMATOUS NASAL SWELLING IN A BOVINE*

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In 1933, Creech and Miller[1] reported for the first time in this country a disease in cattle resembling the nasal granuloma or "snoring disease" prevalent among cattle in India. Although they failed in their experiments to transmit the disease in animals, they nevertheless were of the opinion the disease may be of a parasitic nature. Reports from India indicate that there are two separate diseases of the anterior nares of cattle in that country. While the lesions in both diseases are granulomatous in character, they are distinctly different etiologically. Krishnamurti[2,3] has reported one form of the disease as being caused by a rhinosporidium, while Datta[4] found the type of this disease most prevalent in India to be due to a schistosome. The findings of Datta were confirmed by Malkani,[5] Rao[6] and others. Rao[7,8] also was apparently successful in reproducing the parasitic

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form of the disease in cattle. Dikmans[9] working with material obtained from several American cases of nasal granuloma, observed certain bodies in the nasal secretions which resemble *Rhinosporidium* in their general development, and believes they may have an etiological relation to the disease observed in this country. He also states that they may belong to a fungus closely related to the genus *Rhinosporidium*. Schellner[10] recently reported a case of granulomatous swellings in the nasal, frontal and maxillary sinuses of a horse, in which he found yeast cells present in the involved tissues. He believed these organisms which had the characters of a blastomycete to be the causative agent.

Report of Case

History: A ten-year-old Hereford cow, born and raised in Colorado under semi-range conditions, was presented for slaughter at one of the Denver packing plants, on December 11, 1935. On antemortem inspection, this animal showed a copious discharge from the nose and had difficulty in breathing. The inspector described the breathing as a snoring sound that could

Fig. 1. Lesions in right nostril showing variation in size of nodules.

be heard some distance away. The cow was in fair condition and was slaughtered as a "U. S. Suspect."

Gross pathology: The mucous membrane on both sides of the nasal septum was studded with raised tumorous nodules varying in size, and extending from the anterior nares about seven inches posteriorly (figs. 1 and 2). A number of the nodules were fused into rather large masses. Several small nodules were present on the dorsal medium aspect of the nasal septum, some distance from the marked involved areas. The tissues had a

mottled appearance, with greenish foci predominating. The sinuses were not involved.

Bacteriologic findings: Cover-slip preparations made from macerated material revealed the presence of many small, oval, granular bodies in the tissue. One of the larger lesions was seared over with a hot spatula, and an incision was also made with a hot instrument, taking every precaution to avoid contamination in making cultures. From the center of the lesion, an uncontaminated culture of a fungus was obtained on meat-infusion agar after incubation at 37° C. for 24 hours. The culture was submitted to our Washington laboratory and was identified by Miss Vera K. Charles, of the Bureau of Plant Industry, as a species of *Helminthosporium.* Members of this genus of fungi are quite commonly found on grain and are not know to be pathogenic for animals. The spores of the fungus, when inhaled, are probably deposited on the nasal mucosa and the culture obtained may possibly be a contaminant. Before excluding this fungus as a possible etiological factor, the pathogenicity of the organism should be determined, at least, for laboratory animals.

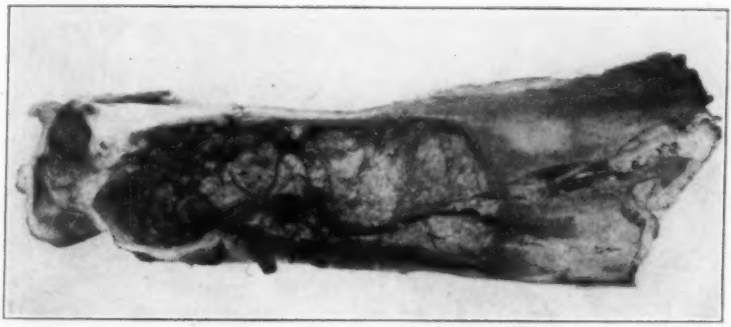

FIG. 2. Lesions in left nostril showing a large, fused mass.

Histologic pathology: Sections made from several nodules and stained with hemotoxylin eosin show a granulomatous structure consisting of numerous blood-vessels, dense masses of eosinophiles, some lymphocytes, plasma cells, a few polymorphonuclear leukocytes and numerous well-formed giant-cells (fig. 3). Within the giant-cells and scattered free in the tissue are seen many spherical bodies with a capsule and containing granular protoplasm. The giant-cells contain from one to six parasites (fig. 4). At the base of the glandular epithelium are seen numerous eosin-staining smaller spherical bodies which, under high magnification, show a capsule and also contain granular protoplasm. We

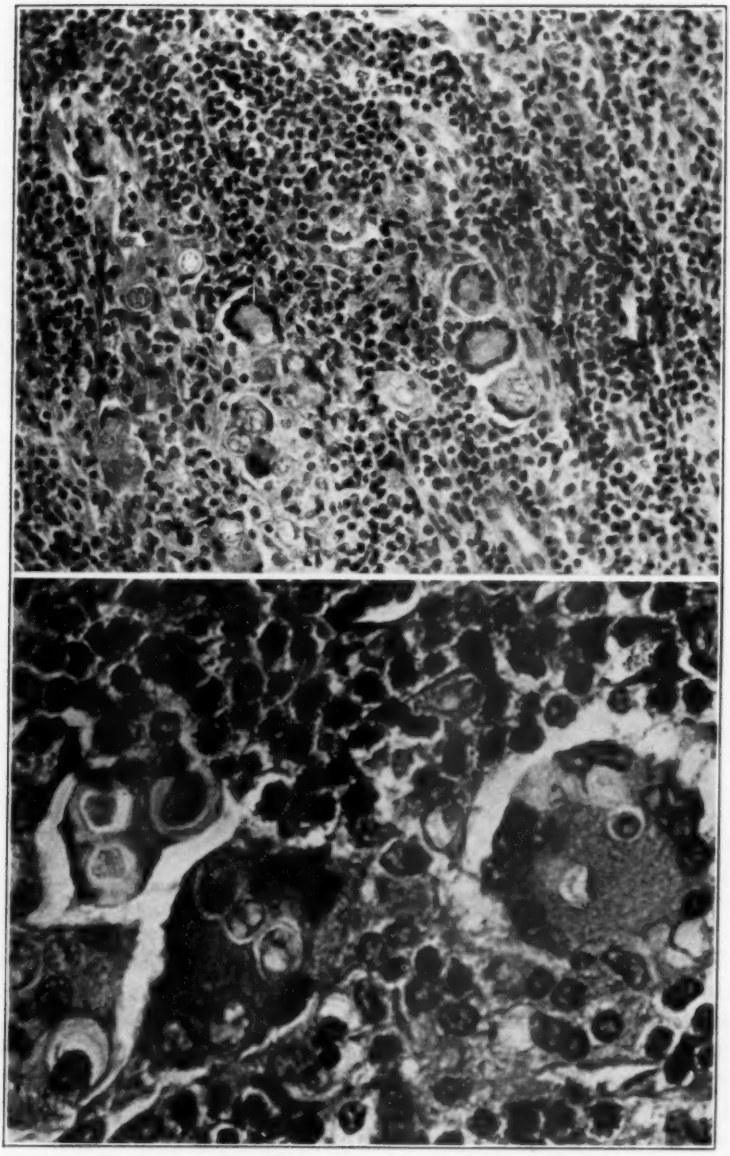

FIG. 3 (above). Section showing several well-formed giant-cells and infiltration with eosinophiles. Note the spherical bodies free in the tissue and within giant-cells (x 225).

FIG. 4 (below). Higher magnification showing five pasasites within a giant-cell (x 650).

believe these bodies to be the younger forms of the parasite. There is degeneration and vacuolization of the glandular epithelium. Eosinophiles have infiltrated many of the mucous glands. There is very little tendency to fibrosis. Suppuration is not seen. Cross-sections of the blood-vessels show an increase of eosinophiles in the blood-stream suggesting eosinophilia. In several of the sections are seen a few rather large hyaline bodies. In one field, there is an actinomycotic-like clump of organisms completely surrounded by giant-cells. The parasites are gram-negative.

Differential diagnosis: The pathology observed in this case must be differentiated from the nasal granuloma described by Creech and Miller, and by Dikmans. Although the clinical aspects of the Colorado disease resemble that of nasal granuloma, histologic sections reveal rather distinct pathological differences. In nasal granuloma, there is much greater tendency toward fibrosis, very few giant-cells, and an absence of spherical bodies seen in our disease. The salient points of differentiation between this case and coccidioidal granuloma in animals is that in the latter disease the cases thus far reported showed lesions only in the bronchial or mediastinal lymph-glands, sometimes in both, with caseation necrosis and from which the fungus coccidioides was recovered. Coccidioidal granuloma is pathogenic for guinea pigs. Also, the coccidioides possesses a distinct double-contoured capsule not seen in the spherical bodies in our case.

Discussion

The failures experienced by investigators in the experimental transmission of nasal granuloma, as it occurs in this country, have rendered it quite difficult to reach any very definite or satisfactory conclusion with regard to the probable nature of the etiological factor involved. Creech and Miller obtained several fungi from their cases of nasal granuloma, and because inoculation experiments with these were unsuccessful they considered their bacteriologic findings entirely negative. There is no doubt in our minds that the spherical bodies present in the tissue in this case are responsible for the lesions. Until the pathogenicity of the recovered fungus is determined, we can look upon this organism only with suspicion as a possible etiological factor. Perhaps we are dealing with a normal contaminant of the nasal mucosa that has assumed pathogenic properties.

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TREMBLING IN PIGS*

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During the spring farrowing season of 1936, two litters of pigs were farrowed in the California Experiment Station herd, individuals in which exhibited a marked trembling. Not all individuals in the litters were affected. One litter consisted of Poland Chinas, the other Duroc Jerseys. In the Poland China litter ten pigs were alive at birth and one was dead. The total birth weight of the litter was 35 pounds. Of the ten pigs born alive, five manifested the condition. Two were affected severely, two moderately, and one only slightly. One of the severely affected pigs shook violently at birth, while the others developed the condition very soon thereafter. The average birth weight of the affected pigs was 3.5 pounds. At ten days of age, two of the pigs (one severely and one moderately affected) were given about 10 cc of calcium gluconate. Two other pigs (one severely and one moderately affected) were given, by mouth, a small dosage of a weak solution of potassium iodide for two consecutive days. Neither of these gave any immediate relief.

A few days later, the two pigs moderately affected and the one pig only slightly affected became normal in so far as outward appearances were concerned. There was no change in the two severely affected pigs. At six weeks of age, they still exhibited some trembling though not so severe as formerly; however, their joints became enlarged. They would lie on their sides away from the other pigs and had difficulty in arising alone. Shortly thereafter, one of the pigs died and the other one was not doing so well as the other pigs in the litter.

There were nine pigs in the Duroc Jersey litter that were

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affected. This litter weighed 23 pounds at birth. This was not a strong litter and all the individual pigs showed a tendency to spraddle behind. Five pigs in this litter exhibited different degrees of trembling. The condition developed soon after birth. The trembling gradually disappeared and could not be detected 14 days after birth.

Description

The shaking and trembling was exhibited only when the pigs were standing. As soon as they would lie down, the trembling ceased except in the case of one of the severely affected Poland China pigs. They shook so severely that every part of the body moved or vibrated and at times their legs would be lifted from the floor. One pig had difficulty in getting a teat in his mouth and one could not walk normally but hopped with his hind legs, both moving forward together. The less severely affected individuals would tremble considerably at times and at other times hardly at all.

Discussion

We believe the dams and sires of these litters are normal hogs. They were raised on normal diets and were fed normal herd rations on green pasture during the breeding, gestation and lactation periods.

The same or similar condition in pigs was noted by Nissley,[1] in Pennsylvania, during the spring of 1932. He reported that more than half the pigs shivered at birth and continued to do so for about two weeks, when it gradually disappeared. Ibsen[2] reports what appears to be a somewhat similar condition in guinea pigs. Tremor in chicks has been discussed by Hutt and Child.[3] They state that congenital tremor in chicks varies from a slight shake to such a severe condition that chicks were unable to stand. They note, also, that the chicks do not shake when squatting at rest. They have shown it to be a hereditary character.

It appears that the condition found in the California Station herd is the same as that reported by Nissley.[1] There is some indication from the description by Hutt and Child[3] that the condition found in the chick and the pig may be the same. Because of the meager data available, no attempt is made to postulate or even guess what the cause of trembling in the pig may be.

References

[1]Nissley, S. M.: Shivers in pigs. Jour. A. V. M. A., lxxxi (1932), n. s. 34 (4), p. 551.

[2]Ibsen, H. L.: Studies in animal reproduction and inheritance. Kans. Agr. Exp. Sta. Biennial Rpt., 1930-32, p. 82.

[3]Hutt, F. B., and Child, G. P.: Congenital tremor in young chicks. Jour. Hered., xxv (1934), pp. 341-350.

NEUTRALIZATION TESTS WITH SERA OF CONVALESCENT OR IMMUNIZED ANIMALS AND THE VIRUSES OF SWINE AND HUMAN INFLUENZA. Thomas Francis, Jr. and Richard E. Shope. Jour. Exp. Med., lxiii (1936), 5, p. 645.

Human and swine influenza viruses were regularly neutralized by their homologous immune sera. However, the sera of animals convalescent from infection with either the swine or human influenza virus possessed little if any neutralizing capacity for the heterologous virus. Hyperimmunization of animals against swine influenza virus tended to increase the neutralizing capacity of their sera for human influenza virus but in an inconstant fashion, whereas repeated inoculations with human influenza virus frequently resulted in sera with strong neutralizing activities against swine influenza virus. Certain antigenic components are possessed in common by the human and swine influenza viruses. The observations serve to emphasize both the immunological distinctiveness and the interrelationships of swine and human influenza viruses.

THE INCIDENCE OF NEUTRALIZING ANTIBODIES FOR SWINE INFLUENZA VIRUS IN THE SERA OF HUMAN BEINGS OF DIFFERENT AGES. Richard E. Shope. Jour. Exp. Med., lxiii (1936), 5, p. 669.

Sera from a high proportion of human adults and new-born infants studied neutralized swine influenza virus, while sera from children under 12 years seldom exerted such an effect. The results of neutralization experiments with human sera and the virus of swine influenza have been compared with the outcome of similar tests with the virus of human influenza, and it seems evident that the presence of antibodies neutralizing swine influenza virus cannot be deemed the result of repeated exposures to the current human type of virus. From the known history of swine influenza and the similarity of its etiologic virus to that obtained from man, it seems likely that the virus of swine influenza is the surviving prototype of the agent primarily re-

sponsible for the great human pandemic of 1918, as suggested by Laidlaw. The presence in human sera of antibodies neutralizing swine influenza virus is believed to indicate a previous immunizing exposure to, or infection with, an influenza virus of the 1918 type.

ACTIVE IMMUNIZATION OF GUINEA PIGS WITH THE VIRUS OF EQUINE ENCEPHALOMYELITIS. II. Immunization with formolized virus. Herald B. Cox and Peter K. Olitsky. Jour. Exp. Med., lxiii (1936), 5, p. 745.

A resistance of high degree may be induced in guinea pigs and mice against experimental equine encephalomyelitis by means of formolized vaccines in which no active virus can be demonstrated. The induced resistance is not due to residual traces of active virus which might have escaped detection in the formolized tissue preparations. Glycerol-treated portions of virus-infected brain did not preserve the antigenic complex. Experimental and epizoölogical findings indicate that equine encephalomyelitis is a blood-borne infection that is transmitted by insect vectors. A method of immunization by means of an active virus is not without danger of setting up foci of infection and spread of the disease; the formolized vaccine removes such a danger.

INFECTION AND AUTOINTOXICATION. Their influence upon hemoglobin production in experimental anemia. F. S. Robscheit and G. H. Whipple. Jour. Exp. Med., lxiii (1936), 5, p. 767.

Infection is often believed to be responsible for anemia. Lack of absorption and definite blood destruction are often believed responsible for the anemia. Accelerated metabolism due to thyroid or dinitrophenol does not modify hemoglobin production in standard anemic dogs. Endometritis lasting over many months will profoundly reduce the production of hemoglobin in the standard anemic dog. A sterile abscess also will diminish the production of new hemoglobin in the anemic dog when liver is being fed but particularly during fasting periods when the usual abundant production of new hemoglobin is reduced to zero. Destruction of red cells and impaired absorption can be excluded as a factor of any significance. The authors conclude that a disturbance of internal metabolism related to hemoglobin building in the body is responsible for the inhibition of hemoglobin production under these conditions.

IMMUNOLOGICAL STUDIES WITH THE VIRUS OF INFECTIOUS LARYNGOTRACHEITIS OF FOWLS USING THE DEVELOPING EGG TECHNIC. F. M. Burnet. Jour. Exp. Med., lxiii (1936), 5, p. 685.

The technic of chorioallantoic membrane inoculation was applied to the study of the virus of infectious laryngotracheitis as it occurs among Australian poultry. When suitably diluted suspensions of virus were inoculated, isolated foci or pocks appear whose macroscopic form and histological structure is characteristic. The number of these foci may be used as a measure of the amount of virus present. Two distinct types of foci were produced by laryngotracheitis strains, one being characteristic of epizoötic strains from New South Wales and from America, the other a Victorian strain which is of low virulence for fowls. No qualitative antigenic differences can be detected amongst these strains but the epizoötic strains are more readily neutralized by immune serum than the enzoötic Victorian strain.

A study of the inactivation of the virus by immune serum shows that (1) the process of inactivation requires time for its completion *in vitro;* (2) the proportionate reduction in titre produced by a given concentration of antiserum is independent of the initial virus concentration; (3) reactivation by dilution is readily demonstrable; (4) virus in the presence of small concentrations of immune serum producing only a slight inactivating effect is rendered incapable of passing a membrane normally permeable to it; (5) the foci produced from partially neutralized virus suspensions are smaller than normal, suggesting delay in the initiation of foci. These findings bring the neutralization of a typical virus by immune serum completely into line with the phage-antiphage reaction as described by Andrewes and Elford.

STUDIES OF A PARALYSIS SYNDROME PRODUCED IN RABBITS AND GUINEA PIGS BY EXTRACTS OF NORMAL PRIMATE BONE MARROW. Raymond A. Kelser and Lester S. King. Amer. Jour. Path., xii (1936), 3, p. 317.

The paralysis-producing principle present in normal bone-marrow, spleen and leukocytes of man and monkeys is not a virus or other type of living agent and is capable of producing a transmissible disease. The disease-producing agent is of a chemical or enzymatic nature, apparently of leukocytic origin. Extracts of high potency for rodents can be regularly prepared from the bone-marrow of man and monkeys, that from monkeys being especially active. Extracts prepared from spleen tissue

are not so satisfactory. Lymph-glands from cases of Hodgkin's disease and spleen tissue from leukemia cases produce highly potent extracts. Both rabbits and guinea pigs are readily susceptible to the intracerebral injection of these extracts. Monkey bone-marrow injected intracerebrally into monkeys does not produce disease, hence it is evident that autoinfection does not occur. Extracts prepared from bone-marrow of rabbit, guinea pig and horse failed to produce disease when injected into rabbits and guinea pigs. Homologous serum did not neutralize the active principle in bone-marrow extracts. The pathologic lesions are discussed.

A Virus Disease of Owls. R. G. Green and J. E. Shillinger. Amer. Jour. Path., xii (1936), 3, p. 405.

A virus disease has been recognized as a natural disease in the great horned owl (*Bubo virginianus*). The infection has been transmitted experimentally to the same species and to the screech owl (*Otus asio*) but not to the barred owl (*Strix varia varia*). The disease is characterized by the formation of multiple abscesses in the liver and less consistently in the spleen. Large intranuclear inclusion bodies occur typically in the hepatic cells. The various stages of formation of these bodies are described.

Effects of Proteins on Electrophoretic Mobility and Sedimentation Velocity of Red Blood Cells. B. R. Monaghan and H. L. White. Jour. Physiol., xix (1936), 5, p. 715.

The isolectric point of normal red cells cannot be measured but is certainly lower than that of any plasma protein. Red cells are easily damaged so that they will absorb protein from low concentrations. Normal red cells do not absorb protein even from concentrated solutions, as is evidenced by the finding that the ratio of the mobility of the cells to that of the proteins themselves is at least as high as in concentrated casein, albumin, gelatin or fibrinogen solutions as in dilute. The finding that the observed mobility of red cells is unchanged or only slightly decreased when bulk velocity is increased by added protein is interpreted as indicating that the red cell surfaces are hydrated. The aggregating effect of certain proteins has been determined and is assumed to be due to their dehydrating effect upon the cells. Some types of cells as beef are not aggregated, as they are resistant to this dehydrating effect. The difference in the

behavior of different types of red cells demonstrates the importance of the nature of the cell as well as of the medium in determining the rate of aggregation and therefore of sedimentation.

THE BACTERICIDAL EFFECT OF ULTRAVIOLET RADIATION ON ESCHERICHIA COLI IN LIQUID SUSPENSIONS. Alexander Hollander and Walter D. Claus. Jour. Physiol., xix (1936), 5, p. 753.

Washed *Escherichia coli* in liquid suspension require a smaller lethal dose than unwashed bacteria; young cultures required a higher lethal dose than old ones. The effect of ultraviolet radiation inactivation of bacterial cells has been attributed to the action of radiation on the suspending material on the cell wall, on enzymes in organisms, on the colloidal structure of the cytoplasm, and on nuclear material or material which controls cell division.

THE DISTRIBUTION AND STORAGE OF FLUORINE IN THE TISSUES OF THE LAYING HEN. Kenneth Haman, Paul H. Phillips and J. G. Halpin. Poultry Sci., xv (1936), p. 154.

All normal tissues of the laying hen, with the possible exceptions of washed feathers and thyroid, contain small amounts of fluorine. Bones have the highest fluorine content, while the more active tissues such as the kidneys and liver are much lower. Muscles have a normal content of fluorine below that of the mentioned internal organs and fat is very low in fluorine. Additions of raw rock phosphate or phosphatic limestone supplements to the diets of laying hens cause increased storage of fluorine in the tissues. The addition of 0.1056 per cent fluorine fed as raw rock phosphate supplement increased the storage of fluorine 13 to 14 times the normal in the bone and from two to three times the normal in the soft tissues.

A FILTRABLE VIRUS, DISTINCT FROM THAT OF LARYNGOTRACHEITIS, THE CAUSE OF A RESPIRATORY DISEASE OF CHICKS. J. R. Beach and O. W. Schalm. Poultry Sci., xv (1936), p. 199.

Experiments have shown that nasal, tracheal and bronchial exudate from two distinct strains of a respiratory disease of chicks which clinically resembles infectious laryngotracheitis when suspended in boullion and passed through Berkefeld V, N and W filters will produce the disease. The results demonstrate the disease is caused by a filtrable virus which passed through

all grades of Berkefeld filters. It was found that chickens that recovered from an infection with one of the two strains of virus were refractory to further infection by either strain. It was also found that the sera from chickens that had recovered from an infection with one strain of the virus would neutralize virus of either strain. Chickens that were refractory to infection with this virus were shown to be susceptible to coryza caused by *Hemophilus gallinarum* and to the virus of infectious laryngotracheitis. Chickens refractory to the latter virus were susceptible to the former. The virus dried by Swift's method and stored in the refrigerator 180 days produced disease. In 50 per cent glycerol and stored in the refrigerator, it has remained alive for 80 days.

THE TRANSMISSION OF FOWL PARALYSIS (LYMPHOMATOSIS). E. M. Gildow, J. K. Williams and C. E. Lampman. Poultry Sci., xv (1936), p. 244.

Fowl paralysis is readily transmitted by pen contact from infected to non-infected chicks. The disease develops to a less extent in chicks from an affected flock than chicks from a non-affected flock when brooded together. The disease is more prevalent in chicks from pullet breeding stock than in those from hen breeding stock from the same affected flock. Evidence of variation in inherited resistance is apparent in different families. No major variation in the incidence of the disease was shown between range-raised and confined-raised birds.

EFFECT OF THE CHEMICAL CONSTITUTION OF SOAPS UPON THEIR GERMICIDAL PROPERTIES. Milward Bayliss. Jour. Bact., xxxi (1936), 5, p. 489.

Pneumococci are especially susceptible to the action of certain unsaturated soaps such as sodium oleate, linoleate, linolenate and clupanodonate. The other unsaturated soaps as well as the hydroxylated and saturated soaps are less effective in killing this organism. The bile salts and sodium salicylate require high concentrations to kill pneumococci. The sulfate ester salts are pneumococcidal in concentrations of 0.01 per cent. *Streptococcus lactis* is more resistant to the action of soaps than the pneumococcus. The effectiveness of soaps in killing the latter micro-organisms does not parallel exactly its effectiveness against the former. The two saturated soaps, sodium myristate and laurate, the unsaturated soaps, sodium oleate, linoleate, linolenate, clup-

anodonate, ricinoleate and abietate. as well as the two sulfate esters, are very effective in killing this streptococcus. Of these compounds only sodium diiodosalicylate and undecylenate are effective germicides in a concentration of 1 per cent at a pH of 8.0 using *Escherichia coli* as the test organism. Sodium diiodosalicylate and sodium undecylenate are also effective against *Staphylococcus aureus* at a pH of 8.0. Sodium abietate is effective above a pH of 8.5.

U. S. Bureau of Animal Industry Adds New Division

A new research unit known as the Animal Nutrition Division has been announced by the Bureau of Animal Industry of the U. S. Department of Agriculture, effective May 1. Dr. Paul E. Howe, since 1924, senior biochemist in charge of nutrition investigations in the Animal Husbandry Division of the Bureau, has been named chief of the new division. He has also been named an Assistant Chief of the Bureau.

According to officials of the Department of Agriculture, the reorganization has been made in order to increase the efficiency on research dealing with fundamental problems of feeding and nutrition of farm animals. The new division will use existing personnel and research facilities to a large extent, in addition to increased laboratory space and equipment made available by the current building program at the National Agricultural Research Center located at Beltsville, Md.

Dr. Howe plans to organize the work of the new division into four sections: One devoted to biological studies of nutritional problems with laboratory animals; another to the biochemistry and physiology of digestion; a third to the nutritive requirements of cattle, sheep, swine, goats, horses and dogs, and general studies of the digestibility of feeds; and the fourth to poultry nutrition.

PERSONALS

Dr. C. D. Chase (K. S. C. '36) has opened an office at Portland, Mich.

Dr. O. A. Meyer (Chi. '11) was appointed City Veterinarian of Alton, Ill., recently.

Dr. J. J. Metz, Jr. (U. P. '36) is assisting Dr. M. P. Fuller (Cin. '19), of Berea, Ohio.

Dr. Delbert Doudna (O. S. U. '36) has been assisting Dr. L. Dunlap (O. S. U. '33), of Zanesville, Ohio.

Dr. W. A. Lawrence (Colo. '36) has accepted a position with Dr. S. R. Espy (Ind. '15), of Oklahoma City, Okla.

Regular Army

So much of S. O. 38 as assigns Major Charles O. Grace to duty at Lexington, Ky., is revoked. Major Grace is relieved from further assignment and duty at Fort Benning, Ga., and directed to proceed to Fort Reno, Okla., and report not later than June 30, 1936, to the commanding officer, Reno Quartermaster Depot, for duty.

Major Raymond T. Seymour is relieved from assignment and duty at the Robinson Quartermaster Depot, Fort Robinson, Neb., effective on or about August 1, 1936, and will proceed to Madison Barracks, N. Y., for duty.

Colonel John A. McKinnon is relieved from further assignment and duty at Fort Riley, Kan., effective on or about August 1, 1936, and will then proceed to Fort Bliss, Tex., and report to the commanding general for duty.

Colonel Burton A. Seeley is relieved from duty at Fort McPherson, Ga., and additional duty at headquarters, Fourth Corps Area, Atlanta, Ga., and will report to the commanding general, Fourth Corps Area, Atlanta, Ga., for duty with the Veterinary Corps at his headquarters, and additional duty as attending veterinarian, Fort McPherson, Ga.

Major George J. Rife is relieved from further assignment and duty at Fort Benning, Ga., effective on or about August 15, 1936, will then proceed to Fort Leavenworth, Kan., and report to the commanding general, for duty.

Lt. Colonel Jacob E. Behney is relieved from duty at Fort Myer, Va., effective on or about August 1, 1936, will then proceed to Fort Riley, Kan., for duty.

Major James R. Sperry is relieved from duty at Madison Barracks, N. Y., effective on or about August 1, 1936, will then proceed to Fort Myer, Va., and report for duty.

Major Seth C. Dildine is relieved from assignment and duty in the office of the Surgeon General, Washington, D. C., to take effect on August 24, 1936, is then assigned to station at the New York port of embarkation, Brooklyn, N. Y., will proceed to Carlisle Barracks, Pa., and report to the commanding general for temporary duty until about December 19, 1936, and will then proceed to Brooklyn, and report for duty accordingly.

Lt. Colonel Clifford C. Whitney is relieved from duty at Fort Clark, Tex., effective at such time as will enable him to proceed to San Francisco, Calif., and sail on transport scheduled to leave that port on or about August 22, 1936, for the Panama Canal Department for duty with the Veterinary Corps.

Major Raymond I. Lovell is assigned to duty at Vancouver Barracks, Wash., effective upon completion of his present tour of foreign service.

The appointment of First Lieutenant Walter Tederoff Carll, Veterinary Corps Reserve, as first lieutenant in the Veterinary Corps, Regular Army, with rank from May 22, 1936, is announced. He will report to the commanding officer, New York port of embarkation, Brooklyn, N. Y., for assignment to duty.

Veterinary Reserve Corps

NEW ACCEPTANCES

Dickman, Andrew Jackson....Capt...727 S. Virginia St., Reno, Nev.
Bridges, Buford Forrest....1st Lt...Route 1, Doerun, Ga.
Schneider, Jr., Morris David.1st Lt...2616 N. 29th St., Philadelphia, Pa.
Willers, Karl Harry.....1st Lt...1028 Greenwood Ave., Canon City, Colo.

PROMOTIONS

To

Boley, Loyd Edwin.....1st Lt...1222 2nd Ave., S. E., Cedar Rapids, Iowa.
Brandly, Paul Jacob.....1st Lt...448 W. 57th St., New York, N. Y.
Burstein, Harold .....1st Lt...1961 N. 31st St., Philadelphia, Pa.
Calldemeier, Huston Adrain.1st Lt...409 S. Jackson St., Louisville, Ky.
Daughtrey, Franklin Delaware .....1st Lt...355 E. 5th Ave., Lancaster, Ohio.
Gatbonton, Alfredo Reyes...1st Lt...Diklom Ranch, Bukidnon, P. I.
Hamilton, Fred Carlisle....1st Lt...6214 Carrollton Ave., Indianapolis, Ind.
Hernandez, Jose Constantino .....1st Lt...1605 Sande St., Tondon, Manila, P. I.
Hesse, Charles Perry.....1st Lt...113 E. 9th Ave., Columbus, Ohio.
Hudson, Claude .....1st Lt...Care Dr. Ralph Graham, 202 Federal Bldg., Jefferson City, Mo.
Johnson, Sanford Edwin....1st Lt...2724 Spaulding St., Omaha, Neb.
Kelsall, III, Samuel.....1st Lt...Box 1086, Sacramento, Calif.
Knapp, John Harold.....1st Lt...146 W. Frambes Ave., Columbus, Ohio.
Larson, Emil Vernon.....1st Lt...Evansville, Minn.
Lee, Chester Daniel.....Capt...1120 Harding St., Ames, Iowa.
Merchant, Willard Roosevelt.1st Lt...6019 Kimbark Ave., Chicago, Ill.
Murphy, James MacMillan..1st Lt...301 S. 43rd St., Philadelphia, Pa.
Nieberding, Joseph Fedelis..1st Lt...506 I. St., S. E., Miami, Okla.
Rash, Ed Ellis.....1st Lt...330 Federal Bldg., Madison, Wis.
Richter, Jacob Benjamin....1st Lt...313 S. 6th St., Philadelphia, Pa.
Rooks, Elmer Lyle.....1st Lt...343 N. 5th St., Greenfield, Ohio.
Sadler, Kenneth Earl.....1st Lt...Wagner, S. Dak.
Schmidt, Clarence Peter....1st Lt...218 Federal Bldg., Sacramento, Calif.
Simonson, Earl Matzen.....1st Lt...Hooper, Neb.
Stults, Amos Waters.....1st Lt...9 E. Broad St., Hopewell, N. J.
Watson, Clement Earl.....1st Lt...1130 Franklin Ave., Lexington, Mo.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Deal, Alfred F.....1st Lt...Fort Totten, N. Y.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Carll, Walter T.....1st Lt...N. Y. Port of Embarkation, Brooklyn, N. Y. (Appointed 1st Lt. in Regular Army)

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

COMMENCEMENTS

A. AND M. COLLEGE OF TEXAS

The sixtieth annual commencement exercises of the Agricultural and Mechanical College of Texas were held at College Station, May 29, 1936. Twelve students in the School of Veterinary Medicine were awarded their degrees, as reported by Dean Francis:

Carlos Juan Cardona
Charles Leonard Coleman
Moray Claude Coop
George Tyson Edds
Ralph G. Gomez, Jr.
Bernard Farr Mood
Philip Myers
Erling Richard Quortrup
George Clinton Rich
Charles J. Steeger
Robert Leslie Sudheimer
Alfred Charles Zedlitz

E. W. Ramsey received his degree at the end of the first term, February 1, 1936.

COLORADO STATE COLLEGE

The fifty-fourth annual commencement exercises of Colorado State College were held June 4, 1936. The Division of Veterinary Medicine presented 17 candidates for the degree, Doctor of Veterinary Medicine:

Wayne Allen Anderson
John Marshall Broadwell
Elvin George Cole
Russell Lee Cowen
Albert Downing Glover
Kenneth Jack Hester
Leslie Wakelin Jones
William Davis Kinchelow
Earl G. Kingdon
Walter Andrew Lawrence
Harry Alfred Leonard
Ernest Henry Leonhard
Frank Harold Olvey
John William Smith
Thomas William Tamoglia
Thomas Edmond Utley
Virgil Addison Yount

Scholastic honors were won by Walter A. Lawrence. With the exceptions of Ernest H. Leonhard, Thomas W. Tamoglia and Virgil A. Yount, who had previously received commissions in the National Guard, and Earl G. Kingdon, all the members of the graduating class were commissioned as First Lieutenants in the Veterinary Reserve Corps of the United States Army, according to Dean Newsom.

J. Pingree Crawford received his degree at the summer commencement in 1935.

STATE COLLEGE OF WASHINGTON

The annual commencement exercises of the State College of Washington were held on June 10, 1935. Thirteen students from the College of Veterinary Medicine were awarded their degrees:

Daniel P. Arron
Emmett Wesley Cantrall
Burton Chapman Christopher
William Paul Guard
Ernest L. Henkel
William Raymond Joneschild
Fleetwood Raymond Koutz
James J. Miller
George Frederick Motteler
Robert Talcott
George Agusta Williams
Henry F. Winiecki
James Winston

Ray Koutz was awarded his diploma with highest honors; George Williams and Emmett Cantrall with high honors, and Henry Winiecki with honor. Ray Koutz also received the set of Proceedings of the Twelfth International Veterinary Congress, given to the senior student with the highest scholastic average.

Ten of the graduates received commissions as First Lieutenants in the Veterinary Reserve Corps of the United States Army.

Karl Bissell and William Jones received their degrees at the end of the summer session, August, 1935, and Harold Peterson received his degree in February, 1936.

Twelve members of the graduating class immediately filed their applications for membership in the A. V. M. A., following a suggestion from Dean Wegner.

UNIVERSITY OF PENNSYLVANIA

The 180th annual commencement exercises of the University of Pennsylvania were held on June 10, 1936. On that occasion, 33 students received the degree, Doctor of Veterinary Medicine, as follows:

Constantine Francis Batory
Hans Baumgartner
Henry Robert Becker
Myron Paul Berk
Earl Wesley Cook
Robert Russell Crelin
John Joseph Devine
Chester Francis Diehl
*Frank Galloway DuBuy
George Russell Flack
Morton Gradess
*Fred Lewis Herring
Charles Patrick Hines
Sperry Clark Kinton
*Donald Gifford Lee
Donald Russell Lundy
Thomas James McConaghy
Russell Howard McCullough
John James Metz, Jr.
Bernard Meyerowitz
Robert Franklin Oplinger
Albert Lebo Opp
Leonard Harold Sherman
Anthony Michael Stefanski
Sidney Alexander Steiner
Russell Francis Strasburger
Henry Sussman
Kenneth Joseph Thomas
Edward Leon Vail
John William Walker
Cornelius Cloete Wessels
Fred John Wigderson
Morris Lionel Ziskind

*With honors.

Dean Dick reports that Dr. Cornelius C. Wessels, who received his degree with the class, came from Smithfield, O. F. S., Union of South Africa, and was graduated from the Veterinary School at the University of Pretoria, Transvaal, South Africa, before coming to the University of Pennsylvania for special work. Richard A. Huebner received his degree on February 15, 1936.

The J. B. Lippincott Prize of $100, for the highest general average in examinations, and the T. E. Munce Prize of $25, for the highest average in the courses in animal industry, were awarded to Fred L. Herring.

The Jeannette Blair Prize of $50, for the best work done in the Small-Animal Clinic, was awarded to Frank G. DuBuy.

The Leonard Pearson Prize, to the member of the senior class, who, in the judgment of the faculty of the School of Veterinary Medicine, was best qualified to receive it, was won by Donald G. Lee.

The 1930 Class Prize of a book, to the member of the senior class for the highest average in the courses in surgery, was awarded to Donald G. Lee.

The Suburban Cattle Breeders' Association Prize of $25, to the member of the sophomore class who attained the highest average in the course of physiology, was awarded to Richard G. Buckingham.

The Anatomy Prize of the Class of 1926, to the member of the sophomore class for the best work done in the courses in anatomy, was awarded to Philip L. Gauntt, Jr.

MICHIGAN STATE COLLEGE

The seventy-eighth annual commencement exercises of Michigan State College were held June 15, 1936. On that occasion, the degree, Doctor of Veterinary Medicine, was conferred on 21 candidates presented by Dean Giltner, of the Division of Veterinary Science, as follows:

Carl Ralph Benton
William Stowell Britton
Norman Gustave Claus
Harold Leon Decker
Charles W. DeLand
Woodrow Alfred Deppa
Samuel Sylvester Fisher
Keith Frick
Robert Thomas Habermann
Werner Fredrick Hillstrom
Charles Patrick Hodder
Frank Leroy Meyer
Warren David Murdock
Otis John Robinson
Roland Orno Scott
Rolland Slaughenhaupt
Robert K. Somers
Karl LeRoy Sutton
Adolph John Sypien
Robert Frank Willson
Francis Hugh Wilson

The Michigan State Veterinary Medical Association Prize of $25, awarded to the senior student with the best record made during his course, was won by Norman Gustave Claus, who also received the set of the Proceedings of the Twelfth International Veterinary Congress.

Arthur Lawrence Green was awarded the Veterinary Faculty Prize of $25, pledged annually to the veterinary sophomore who has made the best scholastic record during his freshman year.

Stephen W. Lange received his degree June 10, 1935, and Lawrence B. Kemp and John C. Schwabland were graduated at the close of the summer session of 1935.

IOWA STATE COLLEGE

The annual commencement exercises of the Iowa State College were held June 15, 1936. Thirty-one students in the Division of Veterinary Medicine were awarded the degree, Doctor of Veterinary Medicine, as follows:

*Robert Louis Alkire
*Robert James Brown
*Clyde Frank Cairy
*Robert David Courter
Arthur Henry Davidson
*John Michael Dermody
William Tillman Dunn
*Albert Clarence Emminger
Charles Raymond Flickinger
*Lloyd Alson Frazer
James Lane Gibbons
William Carrol Glenney
Leon Webb Haggard
Harry Martin Halverson
*Glenn Carlos Holm
*Marvin Henry Jacobs
Max Edward Landsberg
*Armand Arthur Legner
*Howard Elbert McCutchan
*Garrett Hillery McNay
Lawrence Henry Pint
*James William Pirie
Roy Alonzo Resseguie
*Martin George Schmidt
*Earl Keith Spratt
*Donald Kingston Theophilus
*Norman Miller Twisselmann
*Carl Ebert Venzke
Herbert Peter Wessels
Ralph Leland West, Jr.
Frank Burdette Wilkinson

Norman M. Twisselmann was the honor student of the class and the winner of the George Judisch Scholarship Prize, which consists of the initiation fee and membership dues in the A. V. M. A. for five years.

Winners of the G. G. Graham Prizes, awarded to outstanding students in clinical medicine, were Donald K. Theophilus, first, and Lawrence H. Pint, second.

Eighteen of the graduating class (indicated by *) received commissions in the Veterinary Reserve Corps of the United States Army.

OHIO STATE UNIVERSITY

The fifty-ninth annual commencement exercises of the Ohio State University were held on June 15, 1936. The commencement

address was delivered by Dr. George W. Rightmire, president of the University. The College of Veterinary Medicine presented 55 candidates for the degree, Doctor of Veterinary Medicine, the largest graduating class since 1917, as follows:

Fred Ernest Allen
Harvey Baum
Meyer Berliner
William Edward Bills
Donald Herbert Bradley
Floyd Merle Collins
Roger Alward Cook
Donald Elmer Cooperrider
Marion Thomas Cox
Curtis William Cromley
Ida Mae Dodge
Roger Everett Dodge
Delbert Homer Doudna
Robert Watson Dougherty
Harland Freemont Dye
Paul Emerson Eippert
Berton J. Elliott
Harry George Geyer
Alexander Greenfield
Clarence Pierce Hackett
Chester Nahum Harden
Leslie Fredrich Herman
Gerlad William Holmberg
William Sylvanus Houk
James Taylor Johnstone
Kenneth Stanton Jones
Charles Knox Josse
Julius Siland Katz
Burt William Larsen
William Robert Leggett
Donald Miller
John William Mills
Solomon Mirin
Morris Edmund Moore
Herman Leroy Moser
Harold Eugene Moses
Evan Lloyd Moul
Gaylord Jay Nixon
George Frederick Nixon
Vit Hames Novy
John Luther O'Neill
Paul A. Rainey
Wesley Raymond Reed
John William Roscosky
Raymond Vincent Schoentrup
William Carver Schofield
Carleton William Schwiesow
Harry Joel Sinai
John Joseph Solt
Sol Gailen Stephan
William Martin Vogel
Floyd Arthur Vollmer
Robert Phillip Wagers
Wells McCleery Wilson
Vilas Wilford Zuercher

President Rightmire conferred the degrees following the formal presentation of the candidates. The diplomas were awarded by Dean Oscar V. Brumley.

Dr. Ida Mae Dodge is the first woman to receive a veterinary degree from Ohio State University. She met her husband, Dr. Roger Everett Dodge, during their freshman year at Columbus. They were married March 21, 1933, and after that Mrs. Dodge kept house, held a part-time secretarial position on the campus, and maintained an honor standing in her classwork. Both members of the partnership have filed applications for membership in the A. V. M. A. They will engage in practice in Butte, Montana.

Fred E. Allen, highest ranking student in the class, received the set of the Proceedings of the Twelfth International Veterinary Congress. Twenty-one members of the graduating class have filed their applications for membership in the A. V. M. A.

UNIVERSITE DE MONTREAL

Eight graduates from l'École de Médecine Vétérinaire de la Province de Québec, Université de Montréal, received the degree of Doctor of Veterinary Medicine, at the 1936 commencement exercises.

Jos Blanchet
Fernand-Paul Genest
Laurent Michaud
J. P. Morin
Emile Poitras
J. P. E. Rheault
Gérard Roy
Lucien Sylvain

Emile Poitras was awarded a set of the Proceedings of the Twelfth International Veterinary Congress, the special prize offered by the Organizing Committee of the Congress.

CORNELL UNIVERSITY

The sixty-eighth annual commencement exercises of Cornell University were held on June 15, 1936. On that occasion, the New York State Veterinary College presented 23 candidates for the degree, Doctor of Veterinary Medicine, as follows:

Walter Solon Briggs
Robert Frederick Brown
George Edward Burch
Charles Joseph Curtin
Joseph Dufresne
Martin Harold Fremont
Charles Edward Hults
Julia Gertrude Fisher Kinsey
Henry Kriesel
Francois Levesque
Ralph Arnott Maxwell
Clark Allen Metz
Rosario Francis Milici
Sidney Nathanson
Silas Richman
Hyman Sachs
John Chester Sweatman
Irving Wilson Taylor
Frederic Bristol Thomson
John Arthur Ward
Glen John Weaver
Jules Weiner
Walter Johnston Williams

The prizes awarded at the Honor Day exercises, held May 8, were reported in the June issue of the JOURNAL.

Nebraska Clinic in Omaha

The annual clinic of the Nebraska Veterinary Medical Association will be held at Omaha, July 13, beginning at 9 o'clock.

Through the courtesy of the Board of Governors of the Ak-Sar-Ben Live Stock Show, the facilities at Ak-Sar-Ben Field, 63rd and West Center Sts., have been made available for this clinic. Noon lunch will be served on the premises. Following custom, ladies are invited. Special entertainment will be provided. A special invitation will be extended to all visitors to remain for the evening performance of the famous Ak-Sar-Ben Den Show.

The following committee will be in charge: Dr. D. C. Scott, Tekamah; Dr. W. I. Nelson, Herman; Dr. F. C. Ziegenbein, Nebraska City, and Dr. J. H. Copenhaver, Omaha.

Professor Marek Retired

Professor Josef Marek, for many years a member of the faculty of the Royal Hungarian Veterinary College, Budapest, has been retired at his own request. On the occasion of his retirement from active service, a very impressive ceremony was held, at which the dean of the veterinary faculty delivered an address in which he paid his retiring colleague a splendid tribute. Many prominent research men of Hungary were present on this occasion.

In his address, the dean stated that, in the retirement of Prof. Marek, veterinary science suffered an irreparable loss and every effort had been made to persuade him to remain active at least until he should reach the official retirement age. However, displaying the great generosity which had been characteristic of him throughout his active career, Prof. Marek decided to give his eminent pupil, Dr. John Mocsy, the opportunity of taking up his work, with the inspiration of youth and guided by the noble work of his master.

The dean stated further that the importance and reputation of a college depends on its faculty, and Prof. Marek belonged to that group of teachers who recognized that the vocation is fulfilled only if the instructor is inspired and devoted to his work. Prof. Marek always executed his duties with great animation, thoroughness, persistence and admirable energy.

Prof. Marek aimed to create an altar for veterinary science. It is said that the life of a successful teacher is like a candle, the burning of which lights the path of his pupils. This well illustrates Prof. Marek's activities. While teaching, he was an ideal professor and a fatherly and benevolent friend of the students. He descended to the level of his pupils in order to elevate them to his own heights.

He never enveloped his knowledge in mystic darkness and his personality was simple, open and sincere. His broad knowledge, scientific researches and epoch-making works are appreciated not only in Hungary, his native country, but in medical circles abroad. He has received honorary degrees from the universities of

Utrecht and Leipzig, and honorary memberships in numerous scientific organizations, including the American Veterinary Medical Association (1926).

Among his principal scientific achievements may be mentioned his studies on dourine, thrombo-embolic disturbances of the intestine, fowl paralysis, rachitis, therapy of distomatosis, and the use of the stomach-tube and rhinolaryngoscope. With the late Dr. Francis Hutyra he was co-author of "Pathology and Therapeutics of the Diseases of Domestic Animals," the most outstanding work on this subject, which has been translated into six languages. His "Clinical Diagnosis" is a classic and is foremost among veterinary publications. Marek's name will shine among those who are mentioned as examples of serious work and never-relaxing activity. It is hoped that his scientific researches will continue after his retirement.

O. G.

Information Wanted

A roster of veterinarians engaged in public health service is desired by the Committee on Aspects of Veterinary Public Health, of the American Public Health Association. The Year-Book of the A. P. H. A. for 1935-36 carried the report of this Committee presented at the annual meeting held in Milwaukee, October 7, 1935. Dr. R. V. Stone, Director of Laboratories, Los Angeles County Health Department, Hall of Justice, Los Angeles, Calif., is chairman of the Committee, and he requests that all veterinarians engaged in public health work send him the following information:

(1) Name and full address.
(2) Official title and name of public health organization with which connected.
(3) Briefly explain nature of duties such as: milk inspection, meat inspection, rabies control, tuberculosis control, biological production, laboratory diagnosis, etc.

Each veterinarian reporting to Dr. Stone is requested to indicate whether he is a member of the American Public Health Association and his class of membership, such as fellow, active, or life, and the section of the A. P. H. A. with which he is affiliated.

Immediate compliance with the above request is desired upon the part of all veterinarians engaged in public health work throughout the country. In writing to Dr. Stone, give him the names and addresses of veterinarians engaged in public health work who may not see this notice. He will send them questionnaires for reporting the information desired.

Dr. George Hilton Honored

In recognition of his long and distinguished services in the field of veterinary science, the degree of Doctor of Veterinary Medicine *(causa honoris)* was conferred on Dr. George Hilton at the recent commencement exercises of the University of Montreal. Dr. Hilton is Veterinary Director General, Dominion Department of Agriculture, Ottawa, Canada, a position he has held since February 1, 1924. For 14 years he represented Canada on the

DR. GEORGE HILTON

Executive Board of the A. V. M. A. In 1930, while attending the Eleventh International Veterinary Congress in London, he was made an Honorary Associate of the Royal College of Veterinary Surgeons. Last year he represented Canada at Geneva, Switzerland, at a meeting of the League of Nations Committee appointed to consider uniformity in international shipments of meats and meat food products.

Nothing is so firmly believed as what we least know.—MONTAIGNE.

73rd Annual Convention A. V. M. A.
Columbus, Ohio, August 11-12-13-14

SOUTHEASTERN IOWA VETERINARY ASSOCIATION

Twenty veterinary practitioners from ten counties attended the dinner meeting of the Southeastern Iowa Veterinary Association at the Hotel Brazelton, Mount Pleasant, Iowa, April 14, 1936.

A very interesting and instructive program dealing with the spring problems of veterinary practitioners was presented. Dr. H. A. Johnston, U. S. Bureau of Animal Industry, Washington, Iowa, talked on the progress of bovine tuberculosis eradication. Dr. Grant B. Munger, Cedar Rapids, Iowa, spoke on a number of swine problems including hog cholera, anemia of pigs, and crystal-violet hog cholera vaccine.

The following officers were elected: President, Dr. C. E. Hunt, Mount Pleasant; vice-president, Dr. J. Von H. Schantz, Fairfield, and secretary-treasurer, Dr. T. J. McCabe, Mount Pleasant.

P. V. NEUZIL, *Reporter.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The May meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, New York City, on Wednesday evening, May 6, 1936.

Our guest and speaker of the evening was Dr. William H. Dieffenbach, consulting radiologist to Community Hospital in the City of New York, Inc. The topic presented to us by our eminent speaker was "Ultra Short Wave Therapy in the Treatment of Focal Infections and Acute Inflammatory Conditions." As stated in Dr. Dieffenbach's book, "Ultra Short Wave Therapy," this is a new and important discovery in medical history. It has not been fully tried and no one really knows its advantages and limitations, but through the efforts of men like Dr. Dieffenbach, who was the first man in this country to submit to having a radiograph made of a part of his body—his right hand—we will go far in learning of the results possible.

In presenting his subject to us, Dr. Dieffenbach first discussed and then explained the difference between the ordinary ray and

the short wave. He told how radio operators on ships receiving short-wave messages can operate for only about three hours, due to the effects of the current passing through them. Still, these men never seem to be affected by colds. We were then informed of the various wave-lengths, which is a very important feature. If too strong a wave is used, it is possible to burn tissue and destroy it. The favorite length is from 4 to 10 meters. Often a longer wave will produce lesions instead of destroying them. The proper length of short wave will not burn, for it is not applied directly to the skin and does not touch the tissues, but it will pass through the tissues and bone and produce a uniform heat.

Dr. Dieffenbach enumerated the fields in which he had found the ultra short wave valuable. He also stated that it had failed in the treatment of syphilis although it was being used in the treatment of neuro-syphilitic patients with a 30-meter wave and an exposure of three to six hours being given. As previously stated, its main field is in the treatment of acute inflammatory conditions such as carbuncle, sinusitis, tonsillitis, iritis, any acute infection of the larynx, colo-cystitis, gastro-enteritis, pelvic and bladder infections, bone and teeth diseases, and certain skin conditions. It is used in rheumatoid arthritis but is of no avail in deforming arthritis.

The speaker then made comparisons as to conditions in which this treatment could be applied to small and large animals. He stated that although he had had little or no experience in applying this line of treatment to animals, he could see no reason why it could not be adopted and a logical wave-length applied. It is also thought to be practical in arthritis, tendinitis or other acute traumatic or infectious conditions in the horse.

There is much to be learned about this short-wave therapy and Dr. Dieffenbach urged the members of our profession to avail themselves of the opportunities which present themselves in seeking out the practical and applicable uses of this new medical discovery. It is not diathermy, but a short wave producing no heat, and having an effect on tissues and organisms which as yet is still to be enlarged upon.

A rising vote of thanks was tended Dr. Dieffenbach for a most interesting talk and an opportunity of hearing directly from the man who is largely responsible for the work done to date with short-wave therapy. Reading Dr. Dieffenbach's book, "Ultra Short Wave Therapy," will give the interested individual a clearer viewpoint of what is known on this subject.

The membership of this Association stood in silence with bowed

heads, when the president, Dr. C. V. Noback, announced the passing of one of our oldest members, one devoted and active in working for the development of the Association, and one who was highly respected by all his fellowmen—Dr. Thomas E. Smith, of Jersey City, New Jersey.

During the past four years we have been most fortunate in having had the friendship of Lt. Col. C. B. Perkins, V. C., who is stationed at Fort Jay, Governors Island, New York, as the station veterinarian. Presently, orders will send him to the Philippines and we will all miss his cheery greeting which was always given with a story and a smile. Colonel Perkins has done much to make us realize the splendid work the Army veterinarian is carrying on in his branch of veterinary progress and his interest in our Association is known and valued by all our members, and, as one of us, he will be truly missed but never forgotten and we hope that some day soon, orders will return the Colonel to our group again.

R. S. MacKellar, Jr., *Secretary.*

CENTRAL NEW YORK VETERINARY MEDICAL ASSOCIATION

The 27th annual meeting of the Central New York Veterinary Medical Association was held at Genoa, June 3, 1936 in the auditorium of the schoolhouse. There were 25 veterinarians in attendance, consisting of active members, honorary members and visitors.

Dr. Joseph L. Mosher, of Genoa, presided. The minutes of the previous meeting were read and approved. The report of the Treasurer was submitted to the Auditing Committee, approved and ordered placed on file. The Secretary read a communication from the New York State Veterinary Medical Society relative to the proposal of affiliating local veterinary organizations with the State Society. Full details of the plan have not been worked out, however, and after a lengthy discussion, the members voted in favor of the plan, provided that the final details, as worked out, meet with the approval of this association.

The matter of the expense in connection with the malpractice suit of Dr. A. H. Ide, of Lowville, was then brought up by the Secretary. Dr. M. C. Markham, of Binghamton, gave a detailed account of the trial which resulted in a verdict of no cause for action. At a previous meeting, it was unanimously decided that Dr. Ide be reimbursed for the expense of defending the suit. Dr. W. M. Long, of Baldwinsville, moved that each member be

assessed $5.00 to pay the first installment, and that a second assessment be made, if necessary. The total amount to be raised was stated to be $600, this being for the attorney's fee only. Dr. Ide had taken care of all the other costs himself. The motion was carried unanimously. The New York State Veterinary Medical Society will be asked to assume the remaining $400.

A motion prevailed to have each member subscribe $5.00 toward a special fund, such as was done about twelve years ago, these subscriptions to be the nucleus of a fund to be used for one purpose only, namely, for defending a member in some future malpractice suit.

The election of officers for the ensuing year resulted as follows: President, Dr. Frank N. Burke, Syracuse; vice-president, Dr. E. L. Montfort, Morrisville, and secretary-treasurer (re-elected), Dr. W. B. Switzer, Oswego.

As a part of the literary program, Dr. E. L. Brunett, of the New York State Veterinary College, Ithaca, read a paper on fowl-pox. It was very interesting and instructive, and an animated discussion followed. Dr. E. L. Montfort, of Morrisville, related a few case reports from his practice. Such reports are always interesting and bring out discussions and suggestions that help us with our cases. Dr. I. F. Brenning, of Syracuse, read a paper on "Pyometra in the Bitch." This proved to be quite interesting and Dr. H. C. Stephenson, of Ithaca, led the discussion.

Following the adjournment of the meeting, we made our way to the banquet table, where a very pleasant hour was spent. Dr. J. N. Frost, of the New York State Veterinary College, was the speaker of the evening and gave an account of his European trip.

W. B. SWITZER, *Secretary-Treasurer.*

CHICAGO VETERINARY MEDICAL ASSOCIATION

The members of the Chicago Veterinary Medical Association "stepped out" the evening of June 16, 1936, at the College Inn, Hotel Sherman, Chicago. It was the occasion of the annual banquet. In addition to partaking of an excellent dinner, the members and their wives witnessed the spectacular ice carnival and were entertained by George Givot and his radio circus. Following the floor show was a bridge party, at which Dr. Merillat demonstrated some very original bidding.

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NECROLOGY

WILLIAM A. CAWLEY

Dr. William A. Cawley, of Bedford, Pa., died on January 10, 1936, after a long illness. He had been in the employ of the Supplee-Wills-Jones Milk Company since 1925.

Born in Lewisburg, Pa., November 26, 1891, Dr. Cawley attended local schools and Bucknell University. He was graduated from the latter institution in 1915, and several years later entered the University of Pennsylvania. He received his veterinary degree in 1921.

During the World War, he was a member of the First City Troop of Philadelphia, stationed at Camp Hancock, Ga. He later served with the 103rd Trench Mortar Battery in France. Previously, he had served with Troop M of the First Pennsylvania Cavalry on the Mexican border. At the time of his honorable discharge from the Pennsylvania National Guard, he held the commission of first lieutenant.

Dr. Cawley was a member of Epsilon Chapter of the Alpha Psi Fraternity.

R. PAUL DINGMAN

Dr. R. Paul Dingman, of Prophetstown, Ill., died very suddenly at his home on June 19, 1936.

Born at Prophetstown, May 5, 1902, Dr. Dingman attended local grade and high schools before entering the Ontario Veterinary College. Following his graduation in 1927, he returned to Prophetstown and engaged in private practice with his father, Dr. R. G. Dingman (Ont. '94), who died about six years ago.

Dr. Dingman joined the A. V. M. A. in 1929. He was a member of the Twelfth International Veterinary Congress. He was a pigeon fancier and held membership in the National Pigeon Association and the American Carneau Association. He is survived by his widow (née Loretta Beth Jacques), two sons, his mother and one brother, Dr. Lyle M. Dingman (Ont. '30).

W. B. H.

E. P. SMITHERS

Dr. E. P. Smithers, of Evansville, Ind., one of the oldest graduate veterinarians in the country, died April 5, 1936, at the Evansville home of the Little Sisters of the Poor, at the age of 87 years.

Born in Saint Louis, Mo., he moved with his family to Richmond, Ind., as a youth and later took a course in agriculture at Purdue University. He then went to the Ontario Veterinary College and was graduated in 1878. He started practice at Vincennes, Ind., and remained active until about 15 years ago, when he retired and moved to Evansville.

WILLIAM H. DERR

Dr. William H. Derr, of Wooster, Ohio, died at his home, May 5, 1936, shortly after an attack of angina pectoris. He was 63 years old in March.

Born in East Union Township, Dr. Derr spent most of his life in Wayne County, Ohio. Following his graduation from the Ontario Veterinary College, in 1893, he entered practice at Mansfield, Ohio. About 25 years ago, he returned to Wooster and joined his father, the late Dr. W. F. Derr, in practice.

Dr. Derr was a member of the Ohio State Veterinary Medical Association and the Wooster Rotary Club. He is survived by his widow, one son, one brother and one sister, Mrs. J. P. Hutton, of East Lansing, Mich.

W. B. WALLACE

Dr. W. B. Wallace, of Syracuse, Ind., died in the Marion (Ind.) General Hospital, June 13, 1936, of heart trouble. He was ill only a day.

Born in Rochester, Ind., in 1851, Dr. Wallace first took up wood turning as a trade. Later he turned to veterinary medicine and attended the Ontario Veterinary College. Following his graduation in 1887, he located at Marion, Ind. In 1927, he entered a partnership with Dr. G. E. Botkin (Ind. '12). A year and a half later, Dr. Wallace sold his interest to Dr. Botkin and retired.

Dr. Wallace was interested in race horses and owned several. One of them, Margrave, was named national grand champion gentlemen's driving horse at the Madison Square Garden Horse Show in New York City.

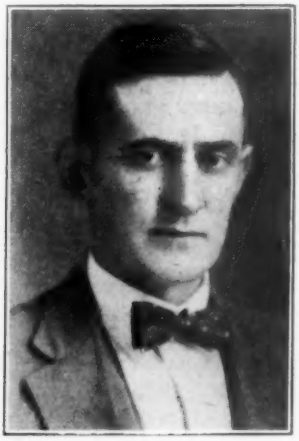
Dr. Wallace joined the A. V. M. A. in 1912. He was a member of the Indiana Veterinary Medical Association. He was a Mason

and an Odd Fellow. His nearest surviving relative is a stepdaughter, with whom he had made his home for two years.

PETER FAIRWEATHER GALLOWAY

Dr. Peter F. Galloway, of Richmond, Va., died suddenly in his office, May 26, 1936, as a result of coronary thrombosis.

Born in Glasgow, Scotland, April 17, 1891, Dr. Galloway was graduated from Queen's Park High School of his native city, and then migrated to the United States at seventeen years of age, locating on a farm in Culpeper County, Va. Three years later, he entered the United States College of Veterinary Surgeons, in Washington, D. C., and was graduated with the class of 1914. Following graduation, he became associated with Dr. Thomas Fraser, of Richmond, Va., and remained with him until the outbreak of the World War.



DR. P. F. GALLOWAY

Dr. Galloway was commissioned as second lieutenant in the Veterinary Corps, June 11, 1917, and the same month reported for duty with the 12th Cavalry at Camp Furlong, N. Mex. He was transferred to the 80th Division at Camp Lee, Va., October 21, 1917. Later he served with the auxiliary remount depots at Camp Hancock, Ga., and Camp Funston, Kans. He was promoted to the rank of captain, January 28, 1918.

Following the war, Dr. Galloway returned to Richmond and formed a partnership with his brother, James, as a result of which the well-known Galloway Brothers Hospital came into

being. He joined the A. V. M. A. in 1915, and was a member of the Virginia State Veterinary Medical Association, and of the Northside Lodge No. 292, A. F. & A. M., and the Scottish Rite bodies.

He is survived by his widow, his mother, three sisters and three brothers.

A. J. S.

JOHN E. WARD

Dr. John E. Ward, of Marquette, Mich., died in Saint Mary's Hospital of that city, June 12, 1936, after an illness of a few days.

The deceased was a graduate of the Department of Veterinary Medicine and Surgery of the Detroit College of Medicine, class of 1896. He practiced in Grand Rapids, Mich., for a number of years, before going to Marquette. He was a member of the Michigan State Veterinary Board about 20 years ago.

Dr. Ward joined the A. V. M. A. in 1912.

JAMES M. McMICHAEL

Dr. James M. McMichael, of Cassopolis, Mich., died at the McCutcheon Hospital in that city, June 15, 1936, after an illness of several months, following a paralytic stroke.

Born in La Grange Township, Cass County, Mich., October 24, 1876, Dr. McMichael attended the Grand Rapids Veterinary College. He was graduated in 1905 and located in Cassopolis, where he practiced until about 20 years ago. During the World War he worked in one of the Dupont ammunition factories in the East. He was unable to enlist on account of club feet.

JOHN P. WILSON

Dr. John P. Wilson, of Hamilton, Ohio, died in Mercy Hospital of that city, June 18, 1936, following a serious illness of two weeks.

Born in Fayette County, Ohio, June 7, 1855, Dr. Wilson attended local schools and then went to New York City to study at the American Veterinary College. Following his graduation in 1885, he entered practice at Hamilton and remained there until his death. He played a prominent part in the development of his city and served as councilman from his ward for one term.

Dr. Wilson was a member of the U. S. V. M. A. from 1885 until 1891.

GEORGE STEPHENS

Dr. George Stephens, of White River Junction, Vt., died at his home, June 12, 1936, of angina pectoris. He had been in a serious condition for over a month.

Born in Turriff, Aberdeen, Scotland, Dr. Stephens came to America and studied veterinary medicine at the American Veterinary College in New York City. Following his graduation in 1897, he located at White River Junction for general practice and remained there until his death. He was a very successful and widely known practitioner and was always interested in everything that was for the betterment of his town and state. In 1903, he was appointed one of the Cattle Commissioners of Vermont and served six years. He was a member of the local school board for 15 years and represented his town in the Vermont Legislature for the session of 1912-13. He introduced a bill which became the Veterinary Practice Act of the state and which created the State Board of Veterinary Registration and Examination. He had served as secretary-treasurer of the Board ever since its organization.

Dr. Stephens joined the A. V. M. A. in 1913. He was a member of the 12th International Veterinary Congress. He helped to organize the Vermont Veterinary Medical Association in 1908 and served as secretary-treasurer from 1915 to 1921, and as president in 1923. A large number of members of the association attended the funeral held at his home, June 14. He is survived by his widow (née Clara Louise Gillette), two sons and a brother.

G. N. W.

EDWARD WILKINS

Dr. Edward Wilkins, of Alexandria, Ind., died in the Saint John Hospital at Anderson, Ind., June 19, 1936. He had been in the hospital for three days and death was due to heart trouble.

Born at Lindsay, Ontario, Canada, August 27, 1865, Dr. Wilkins was a graduate of the Ontario Veterinary College, class of 1891. Before studying veterinary medicine he had been a personal nurse for Walt Whitman, beloved American poet, at Camden, N. J.

Dr. Wilkins practiced in Alexandria for about 45 years and was widely known throughout Madison and adjoining counties. In 1918, he was a candidate in the Republican primary for the nomination of mayor. He served for four years as a member of the City Council. He was an Odd Fellow and served as a trustee of his lodge for more than 20 years.

Surviving Dr. Wilkins are his widow (née Isabelle Hunt), one daughter, two sisters and two brothers.

ELMER B. PARKER

Dr. Elmer B. Parker, of Danville, Ill., died in Saint Anthony's Hospital at Effingham, Ill., June 20, 1936, following an appendectomy three days previously.

Born at Bogota, Ill., March 7, 1894, Dr. Parker attended local schools and then entered the Kansas City Veterinary College. Following his graduation in 1917, he returned to Bogota and entered practice. When the United States entered the World War, Dr. Parker was commissioned in the Veterinary Corps (Oct. 9, 1917) and assigned to duty with the 313th Cavalry at Del Rio, Texas, May 2, 1918. He was transferred to the 67th Field Artillery at Camp Taylor, August 19, 1918; promoted to first lieutenant, Sept. 3, 1918, and discharged on December 23, 1918. He was commissioned in the Reserve Corps as first lieutenant.

After his discharge from military service, Dr. Parker returned to private practice at Louisville, Ill., later going to Newton, Ill. For the past three years he had been in the employ of the Illinois Department of Agriculture, with headquarters in Danville.

Dr. Parker joined the A. V. M. A. in 1917.

ARTHUR W. MUNSON

Dr. Arthur W. Munson, of Bismarck, N. Dak., died at his home, June 23, 1936, of a heart attack. He was born August 21, 1898, at Gladstone, Mich., and was graduated from Ohio State University in 1922. He had been located in practice at Bismarck since his graduation. He is survived by his father, brother and two sisters.

L. M. R.

JACKSON R. BROWN

Dr. Jackson R. Brown, of Geneva, Ill., died in the Evangelical Hospital, Chicago, June 25, 1936, after an illness of six days. He was 52 years of age and a graduate of the McKillip Veterinary College, class of 1908. For a number of years Dr. Brown had been in the employ of the Illinois Department of Agriculture, as supervisor of tuberculosis eradication for the northern part of Illinois, with headquarters at the Union Stock Yards, Chicago.

PERSONALS

MARRIAGES

DR. FRED M. MURDOCK (Mich. '35), of Lansing, Mich., to Miss Ruth Leesley, of Naperville, Ill., at Glencoe, Ill., May 2, 1936.

To DR. and MRS. L. PROCTOR, of Hazleton, Iowa, a daughter, Jananne, May 30, 1936.

To DR. and MRS. S. C. BERNHARDT, of Cassville, Wis., a daughter, Jeanne Ann, June 5, 1936.

BIRTHS

To DR. and MRS. FRANK THORP, JR., of Fort Collins, Colo., a son, Frank Kedzie, April 29, 1936.

To DR. and MRS. P. V. NEUZIL, of Blairstown, Iowa, a daughter, Margaret Anne, April 28, 1936.

To DR. and MRS. R. B. HELMING, of Cresco, Iowa, a son, Robert Hager, March 23, 1936.

DR. LENDALL K. FIRTH (K. S. C. '33) to Miss Josephine Grubb, both of Akron, Ohio, May 16, 1936.

DR. LILLIS R. WEMPE (K. S. C. '35), of Seneca, Kan., to Miss Hazel A. McKibben, of Grantville, Kan., at Manhattan, Kan., May 26, 1936.

DR. F. H. CONOVER (Ont. '34), of New Berlin, Ill., to Miss Freda Arnold, of Jacksonville, Ill., at Jacksonville, June 4, 1936.

DR. L. R. BARTO (U. P. '30) to Miss Marion Miller, both of Summit, N. J., at Summit, June 8, 1936.

PERSONALS

DR. F. M. MARQUIS (San Fran. '14), of San Francisco, Calif., was a hospital patient the early part of May.

DR. GEORGE C. MOODY (Ont. '85), of Mason, Mich., was a hospital patient early in May. He is one of the oldest veterinarians in the United States.

DR. C. H. HOEKSTRA (Chi. '17), of Omaha, Nebr., has been in the Nebraska Methodist Hospital, recuperating from a very serious operation.

DR. A. E. CAMERON (Ont. '08), Chief Veterinary Inspector, Health of Animals Branch, Department of Agriculture, Ottawa, Canada, will go to Europe with the "Vimy Pilgrimage" this summer. He will be away from July 16 to September 10.

DR. J. P. TORREY (Mich. '31) recently resigned his position with the diagnostic laboratory of the Illinois Department of Agriculture, at Urbana, and is now with the Corn Belt Serum Company, of East Saint Louis, Ill., as laboratory and field consultant.

DR. JOSEPH P. SCOTT (O. S. U. '14), of Kansas State College, has been granted a leave of absence, to accept a temporary appointment with the U. S. Bureau of Animal Industry, in a coöperative research project at the School of Veterinary Medicine of the University of Pennsylvania, in Philadelphia.